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United States
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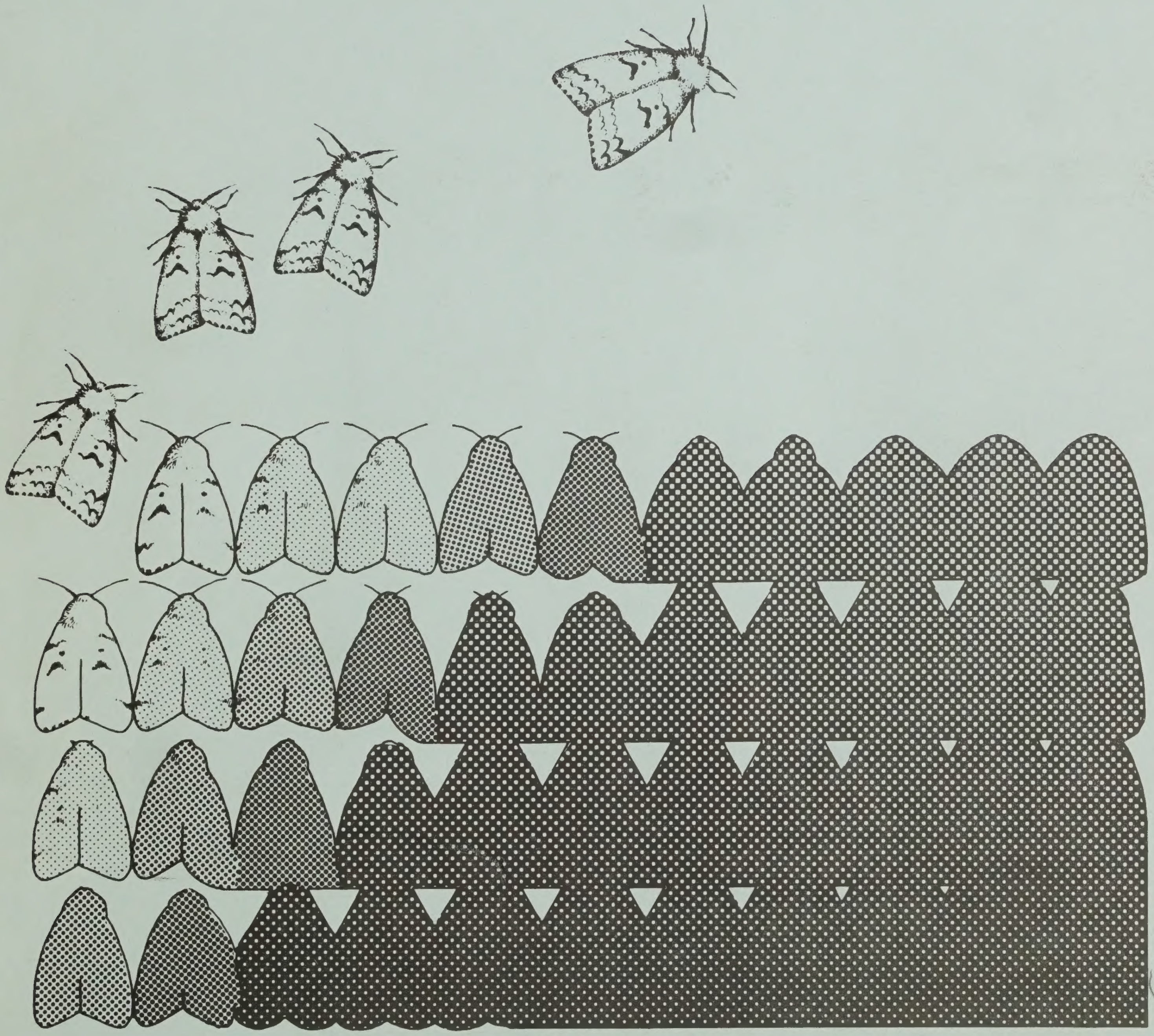
Gypsy Moth Suppression and Eradication Projects

Final Addendum to the Final Environmental Impact Statement as Supplemented - 1985



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Final Addendum to the Final Environmental Impact Statement on Gypsy Moth Suppression and Eradication Projects as Supplemented — 1985

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Abstract: This document provides a plain language version of the worst case analysis in the Final Environmental Impact Statement on Gypsy Moth Suppression and Eradication Projects as Supplemented - 1985 (FEIS). The FEIS was approved on March 8, 1985. Since that date, the United States District Court for the District of Oregon, in Oregon Environmental Council v. Kunzman (Civil No. 82-504-RE), ruled that although the main text of the Final EIS was legally adequate, the worst case analysis in Appendix F failed to meet the regulatory requirement for clarity. In response to this ruling, the Forest Service and the Animal and Plant Health Inspection Service have prepared this addendum. The primary part of this Final Addendum is a plain language version of Appendix F. In addition, toxicity data and cancer risk calculations have been clarified to make the risks more understandable.

This document has been provided to agencies, organizations, and individuals listed in Appendix B of the FEIS, as well as to all those requesting copies. Comment letters on the Draft Addendum and USDA's responses are contained in Appendix J of this document. This addendum shall be used in conjunction with the FEIS when reviewing possible gypsy moth suppression or eradication programs involving USDA agencies or funds.

Date of transmission to EPA and the public: February 12, 1986.

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Introduction

On April 26, 1985, the United States District Court of Oregon ruled that the Final Environmental Impact Statement for Gypsy Moth Suppression and Eradication Projects, as supplemented - 1985 (FEIS), was legally adequate but that the worst case analysis was not understandable by its intended readers. In making this ruling the Court found that although the worst case analysis in Appendix F of the FEIS contained all the necessary information, it was too technical, complex, and full of equations and calculations. This document responds to the Court's ruling.

Appendix H of this document is a plain language version of Appendix F. It translates the technical data contained in Appendix F into terms that disclose possible human health problems in language that all readers can understand. Appendix I includes the following information:

(1) A verbal description of the toxicity tests summarized in Tables 1 through 7 in Appendix F. This description is included to clarify the basis for setting the no-observed-effect levels (NOELs) used in the worst case analysis. It also provides the descriptive background needed to identify possible health effects resulting from exposure to insecticides used to control the gypsy moth.

(2) Clarification and recalculation of the cancer potency and risk of N-nitrosocarbaryl.

(3) Clarification of the cancer potency and risk of 4-chloroaniline, a breakdown product of diflubenzuron.

(4) Clarification of cumulative effects that could result from being exposed to both eradication and suppression projects in a lifetime.

(5) Possible contamination of diflubenzuron with dioxin.

(6) An errata sheet correcting typographical errors in references.

Because this document does not propose substantial changes in the proposed action or contain substantial new information, the Forest Service and the Animal Plant Health Inspection Service (APHIS) do not consider it to be a supplement under the National Environmental Policy Act regulations. This document contains only information that was presented to the U.S. District Court to clarify issues about human health risk and the plain language version of Appendix F.

This document has been provided to agencies, organizations, and individuals listed in Appendix B of the FEIS, as well as to all those requesting copies. Comment letters on the Draft Addendum and USDA responses are contained in Appendix J of this document. This addendum shall be used in conjunction with the FEIS when reviewing possible gypsy moth suppression or eradication programs involving USDA agencies or funds.

Appendix H
Plain Language Summary of the Health Risk Analysis

OVERVIEW

Four chemical insecticides are being considered for use in projects to suppress or eradicate gypsy moths. Appendix F analyzed the risk to human health of using each insecticide. In doing so, it was necessary to use scientific methods, terms, and formulas. Without such scientific rigor, it would be hard, if not impossible, for specialists to judge whether the conclusions are correct.

This appendix is for the general reader. It describes the methods and results of the risk analysis in words that can be understood by decision-makers and the public. The difference between these two appendixes is the level of detail--not the level of accuracy. Readers who want to check the math or see what studies were used should refer to the Final Environmental Impact Statement and Appendix F.

Conclusions

The chemicals being considered are acephate, carbaryl, diflubenzuron, and trichlorfon. The basic question being asked in the risk analysis is, Would human health be affected by their use? Briefly, the answer is as follows:

- All realistic doses to the general public from routine spraying would be unlikely to pose any significant risks of adverse effects, based on evaluations of these chemicals made by the Environmental Protection Agency (EPA) or the World Health Organization.
- All exposures from routine operations would be below levels that could cause birth defects in the general population.
- With two possible exceptions, realistic doses to workers from routine operations would have no ill effects. The two exceptions are mixer/loaders working with acephate and trichlorfon. Where there are effects, they likely would be minor and would not last long. Symptoms might include mild dizziness, headaches, or eye irritation.
- About half the estimated doses from abnormally high exposures (worst case doses) in routine operations

would be above the "safe" levels. That is, they would be above the acceptable daily intake levels. At most, there would be less than 1 chance in 500 of such a high exposure. The effects (if any) would be minor.

- Some people who are unusually sensitive to chemicals could be affected by the routine spraying of any of the four chemicals. These people should be warned of possible harm before spraying takes place.
- In most cases, aircraft spills would have no lasting effects on human health.
- The most severe effects could come from truck spills. Symptoms could range from nausea to shortness of breath to death. With prompt medical help, most symptoms can be reversed. Moreover, the odds of a truck spill occurring are very low. Such a spill probably would occur less than once for every 100 million acres treated. (That is about 250 times the number of acres that have been treated each year in the past.)
- The odds of a person getting cancer from routine operations are estimated to be 4 in a million. This is about the same risk as smoking eight cigarettes in an entire lifetime. In most cases, the odds would be much lower.
- The total added risk of cancer from routine operations would be about 0.05 incidence per year in the exposed population of 5.4 million people. This figure is based on the amounts that have been sprayed in the past.
- It seems extremely unlikely that spraying projects would result in mutations that could be passed to offspring.

Method

These conclusions were reached by using a three-step process used in most risk analyses:

(1) Hazard Identification

- What are the toxic (poisonous) properties of each chemical?
- What doses are deemed safe for humans?
- What doses might cause harm?

Most of this information comes from laboratory tests that used mammals. Other sources include studies of human poisonings and research involving other organisms.

(2) Exposure Analysis

- Who is likely to be exposed as a result of spraying?
- How much of the chemical is likely to enter their bodies?
- How often will they be exposed?

People can be exposed in several ways and can take in different amounts of the chemicals. The exposure analysis describes the ways people might be exposed. These situations, called "scenarios," range from properly handled routine operations, through the worst cases that could occur during routine applications, to accidental spills of chemical concentrates. These scenarios cover a wide range of possible exposures so any real life exposure should be close to or less than these.

(3) Risk Evaluation

- How will human health likely be affected by actual spraying operations?

This is answered by comparing the results of the first two steps. That is, the estimated doses (the amounts that might enter people's bodies) in the different exposure scenarios are compared with the doses found to be safe or harmful to health.

It must be remembered that exposure to insecticides almost always involves some element of risk. Therefore, the risk evaluation discusses degrees of risk (or the odds that significant risks will or will not occur) rather than absolute safety versus unacceptable risks.

Use of Worst Case Assumptions

Whenever there is doubt about what might happen when the chemicals are used, this analysis assumes the worst. For instance, if there is doubt that a chemical can cause cancer, this analysis makes the worst case assumption that it can. Another example is that all standard application rates are increased by 10 percent to account for normal variations in preparing and spraying the chemicals. In the worst case scenarios, these rates are increased by 100 percent to account for possible major errors in mixing and spraying. It is also assumed that a person might eat fruit or vegetables that contain spray residues, even though spraying is done some time before most fruit and vegetables would be harvested. Further, the amount of food in an exposed person's diet is assumed to be greater than it is in the average diet.

Together, the worst case assumptions help ensure that health risks will not be understated. But in doing so, they probably suggest that the spraying projects pose greater risks than are likely. In other words, the risks listed above probably are exaggerated. For example, use of worst case assumptions suggests that there is a small risk of cancer occurring. Yet there is no evidence that any of the four chemicals have caused cancer in humans at any dose levels.

The following sections discuss the methods and conclusions of the health risk analysis in greater detail.

HAZARD IDENTIFICATION

Determining Toxicity

The first step in the risk analysis is to determine the toxicity of each chemical. Toxicity is the ability of a substance to harm health.

All four chemicals have been studied in the laboratory using conventional toxicity tests on animals. It is standard practice in the health field to use the results of such tests to help determine hazards to people. This is because different animals, including humans, often react similarly when given similar doses of a substance. But this is not always the case. For example, dogs seem much more sensitive than other mammals to carbaryl. Therefore, good judgment and care must be used when applying the results of animal tests to humans.

Health effects caused by toxic chemicals fall into two groups: threshold responses and nonthreshold responses. Most obvious effects, such as birth defects and nervous disorders, seem to fall into the first group. Cancer and mutations (changes in body cells) fall into the second group. But it is not the types of diseases that separate the two groups. Rather, it is the way in which the responses occur.

With threshold responses, there is a certain amount of the substance that can enter the bodies of most animals or humans without causing any harm. The dividing line between doses that have no effects and those that do is the threshold level. Once the threshold is crossed, increased doses will increase the intensity and extent of the effects. In theory at least, these types of responses are fairly predictable and similar in all healthy people.

With nonthreshold responses, any dose might set off a reaction, but there is no certainty that it will do so. There will be an adverse effect only if the chemical successfully invades the body and reaches certain strategic points, such as the DNA in human cells. It is possible for a large dose of the substance to enter a person's body without any effect at all. But the greater the lifetime dose, the greater the odds of seeing these effects.

Threshold Responses

Animals and people do not show threshold responses until certain doses are exceeded. Therefore, to assess health risks from a chemical, it would seem necessary to find out its threshold dose. That is, how much of the chemical will the body tolerate before there are ill effects? This threshold dose cannot be known precisely without running a seemingly endless series of tests using slightly different doses. So toxicologists instead focus on the highest doses that are known to cause no ill effects. These doses are called no-observed-effect levels (NOELs).

No-observed-effect levels for chemicals are determined in standard, controlled lab tests. In these tests, a population of animals (such as mice of roughly the same age) is separated into groups. Each group then is given a different daily dose of the substance for an extended period of time. The highest dose that has no apparent ill effects is the NOEL.

To compare doses given to different species or different sized animals, a common unit is needed for measuring doses. Sometimes doses are based on body weight. At other times, doses are based on the surface area of the body. In this study, doses are expressed as fractions of body weight. The standard unit is milligrams (of chemical) per kilogram (of body weight). One kilogram is equal to 2.2 pounds, while a milligram weighs a million times less. When the dose is given daily, as is the case with most NOELs, the unit is milligrams per kilogram per day (mg/kg/day).

There may be several NOELs for each chemical--both for different species and different responses. While different species of mammals, including humans, tend to respond similarly to the same dose of a chemical, they do not respond identically. Furthermore, toxicity tests often look for specific types of responses (such as birth defects) and might overlook others. Figure H-1 shows a hypothetical example of a substance with several NOELs.

This hazard analysis tried to focus on the lowest NOEL for each chemical to make sure that risks would not be understated.

As suggested in Figure H-1, once the dose of a substance exceeds the NOEL and crosses the threshold, the effects tend to increase as the dose increases. The increase in effects can take two forms: an increase in intensity (such as increasing kidney problems), or the addition of new types of effects (for example, birth defects in

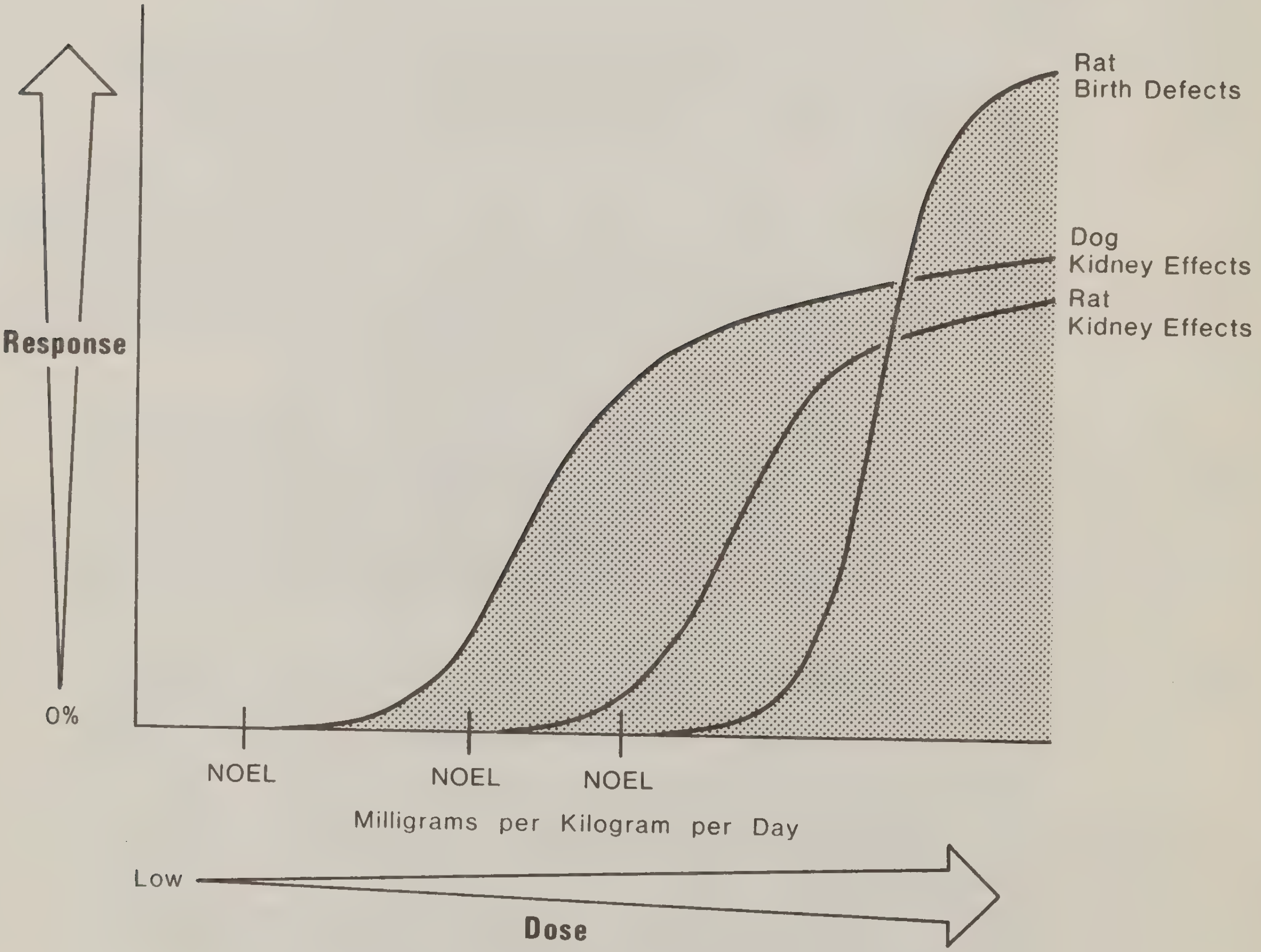

Figure H-1.--Typical dose-response pattern for threshold responses. This example shows a chemical with three no-observed-effect levels (NOELs). The lowest NOEL is for kidney effects in dogs. The intensity of this effect increases as the dose increases. Even larger doses could result in birth defects in addition to kidney problems. (The two NOELs for kidney effects show how one species might be more sensitive than another to the same chemical.)

addition to kidney problems). The first effects might be relatively mild and, even then, result only after long-term exposure. But as the dose increases, the effects would become more severe.

The most severe effect from a toxic substance is death, with the most extreme effect being death from a single (acute) exposure. The one-time or short-term dose that kills 50 percent of a group of treated lab animals is called the LD_{50} (for "lethal dose, 50 percent"). An oral LD_{50} is the lethal dose from swallowing a chemical. A dermal LD_{50} is the lethal dose on unbroken skin. Clearly, a person would be at great risk if exposed to a substance at a level near or above its LD_{50}.

Because of biological differences between test animals and humans, NOELs cannot be responsibly applied to humans without using a safety factor. That is, to err on the side of caution, NOELs from animal studies usually are reduced by a safety factor to set safe doses for humans. The most common safety factor is 100, but it can range from 10 to 1,000. The U.S. Environmental Protection Agency and the World Health Organization both use safety factors to establish safe doses for various chemicals. For each chemical, the safety factor used depends on how sure they are that the available studies can be applied to humans. The term they use for the safe dose is "acceptable daily intake (ADI)." The ADI is believed to be the maximum dose of the chemical that can be taken every day over a person's lifetime without any adverse effects. Figure H-2 shows the relationship between NOELs, ADIs, and LD_{50}s.

ADIs are the starting points used to evaluate the health risks associated with each chemical. If the exposure analysis shows that the expected dose to humans will be less than the ADI, then the dose is considered safe for most people. For doses above the ADI, it is necessary to look more closely at the data about the chemicals. Specifically, it is necessary to determine the margin of safety--that is, to see how close the dose is to the various NOELs. It is also necessary to look at the type of responses that might be involved. If the expected dose occurred every day for a long period and approached the NOEL for a response that is not easily cured, then the safety margin would be small and the health risk might be great. In such situations, a responsible decision-maker would want a high margin of safety before using the chemical.

It must be kept in mind that the NOELs used in this analysis are from studies that involve daily exposure over a long time. Also, ADIs are considered to be doses that can be taken safely every day for an entire lifetime. Yet

LD_{50} - Acute lethal dose. One-time or short-term dose that is lethal to 50 percent of treated animals.

Threshold - Long-term dose level at which adverse effects first occur.

NOEL - No-observed-effect level. Long-term dose that does not result in apparent adverse effects in test animals.

Safety Factor - Factor applied to the NOEL to set safe lifetime dose to humans.

ADI - Acceptable daily intake. Maximum dose that a person could safely take every day throughout lifetime without harm to health.

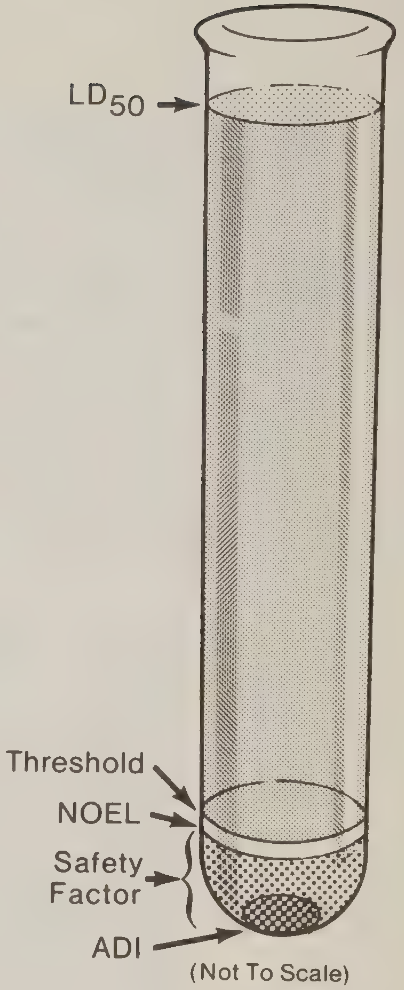

Figure H-2.--Relationship between doses seen in laboratory tests and established safe doses (acceptable daily intakes) for humans.

most potential exposures from gypsy moth projects are one-time or short-term exposures. Therefore, comparing the estimated exposures to ADIs and NOELs might not give a true picture of the risks involved. Any error would overstate the risks.

Nonthreshold Responses

Scientists do not all agree about the link between human exposure to chemicals and the occurrence of mutations or cancer. But generally it is thought that no threshold levels are involved.

Cancer. Doctors usually do not speak of degrees of cancer; a person either has cancer or does not. The chance of getting cancer has been compared to the chance of being hit by a car when crossing a road blindfolded. Even if there is only one car within 100 miles, there is a small chance it will hit. If there are two cars, the chances will be greater, and so on. Likewise, the odds of getting cancer from a known carcinogen (cancer-causing substance) increase with the size and duration of the dose.

Hazard assessments for cancer have two steps. The first is to see if there is evidence that a chemical could cause cancer. The second is to find the odds of getting cancer from different doses. Since there are no known cases of human cancer being caused by any of the four chemicals, data on animals were used. Where tests indicated that a chemical might cause cancer in mammals, a mathematical model was used to determine its cancer potency.

Various models (or formulas) can be used to determine cancer potency. For this study, a simple linear model was used. The linear model assumes that a steady increase in dose will result in a steady increase in the odds of getting cancer. This model is overly simplistic, but it usually errs on the side of overstating the chances of cancer occurring.

The linear model also assumes that a given total dose will have the same effect no matter what the dosing period is. For example, a large dose given on 1 day is assumed to have the same effect as the same total dose given in smaller amounts over several days. However, this may not always be the case. So this assumption puts some uncertainty into the risk analysis.

An example of the assumed linear relationship between a dose of a specific substance and the chance of cancer is shown in Figure H-3. To show how the linear model overstates the effects of low doses, the graph includes a curve that is closer to known cancer potencies.

The straight-line slope in Figure H-3 represents cancer potency. It shows what the increase in cancer probability is for each increase in dose. If the slope were steeper, then the cancer potency would be greater. The potency slope also can be expressed as a number; the higher the number, the more potent the carcinogen. Because potency slopes and values can be difficult to grasp, this summary also indicates what daily dose of each chemical would result in a 1 in a million chance of getting cancer from that chemical.

Heritable Mutations. Cancer is the end result of a multi-step process that starts with mutations (changes) in body cells. Changes in most body cells might lead to cancer. But changes in most cells cannot be inherited by offspring. Cells involved in reproduction--called germ cells--are another matter. Mutations in these cells can be inherited. Some of these changes may be minor, but others can be quite serious.

At this time, there are no generally accepted mathematical models for determining the risk of mutations. Instead,

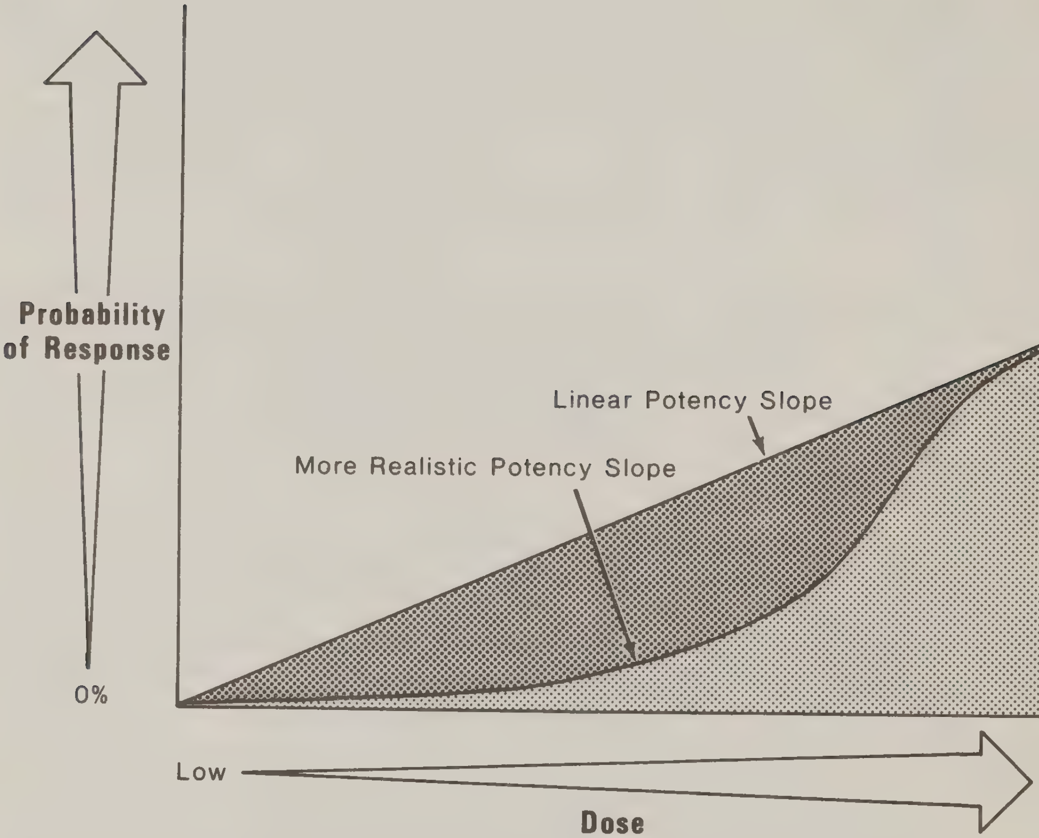

Figure H-3.--Relationship between dose and probability of cancer assumed in linear model. The curved line represents a more realistic potency slope. The darker shaded area suggests how the linear model overstates the effects of low doses.

scientists weigh the evidence from various laboratory tests to try to assess the ability of a chemical to cause mutations in humans. Such a "qualitative" picture is incomplete; it indicates whether the chemical can cause heritable mutations (those that can be passed to offspring), but it cannot quantify the risks for humans.

However, it may be safe to assume that the risk of heritable mutations would be no greater than the risk of cancer. The mechanisms that distinguish cancer development from the development of mutations are not fully understood. Various factors have been identified that could cause cancer or heritable mutations. The main difference is that substances that cause cancer have many more possible targets in the body. While cancer is caused by changes in any cell, heritable mutations are caused only by changes in germ cells. Thus, use of the linear cancer model (which overstates the risk of cancer) to estimate the risk of heritable mutations would grossly overestimate such risks.

Hazard Levels of the Four Insecticides

All the insecticides under consideration are currently registered by the Environmental Protection Agency for the control of gypsy moth larvae. This means that, in EPA's judgment, available studies indicate that none of these chemicals are likely to cause unreasonable adverse effects in people or the environment when properly used.

The acceptable daily intakes, no-observed-effect levels, and acute lethal doses used in Appendix F are compared in Table H-1. The higher the number, the less harmful the chemical. The table also shows the cancer potencies of the four chemicals. For cancer potency, the higher the number, the more harmful the chemical in terms of cancer. The following subsections summarize the toxic properties of each chemical. (More detailed information can be found on pages 42 to 67 of the FEIS, on pages F-12 to F-22 of Appendix F, and on pages I-1 to I-22 of Appendix I.)

Acephate

Threshold Responses. Based on the effects of large, one-time doses, acephate is considered to be a moderately toxic insecticide. Its main health effect is to reduce the active levels of cholinesterase, an enzyme found in red blood cells, blood plasma, and elsewhere and that is essential to the functioning of the nervous system. If this enzyme is bound up by the insecticide (called "cholinesterase inhibition") and thus withheld from the nervous system, a number of ill health effects can occur. The lowest NOELs, which are for cholinesterase inhibition in rats and dogs, are 0.25 milligram per kilogram of body weight per day.

There is no evidence that acephate causes birth defects. However, it can cause maternal toxicity. Symptoms in lab animals have included increased incidences of abortion. The NOEL for maternal toxicity is more than 10 times higher than the lowest NOEL for this chemical. EPA has established the ADI for acephate at 0.025 milligram per kilogram of body weight per day. This means that the highest "safe dose" for a person weighing 154 pounds (70 kilograms) is 1.75 milligrams per day.

Nonthreshold Responses. Acephate can cause gene mutations in cells grown in the laboratory. But these effects have not been seen in whole mammals with intact defense mechanisms.

It is uncertain if acephate can cause cancer. But acephate is suspect since very high dose levels have caused tumors in female mice. Therefore, this analysis makes the worst case assumption that acephate can cause cancer. Assuming this, and using the linear model, acephate's cancer potency

Table H-1.--Comparison of no-observed-effect levels, acceptable daily intakes, acute lethal doses, and cancer potencies of the four insecticides used in the risk analysis

Benchmark	Acephate	Carbaryl	Diflubenzuron	Trichlorfon
	Milligrams per kilogram of body weight			
Dermal LD_{50}	10,000[a]	9,600[a]	2,000[a]	2,100[a]
	Milligrams per kilogram of body weight per day			
Lowest NOEL	0.25	3.125	1.1[a]	1.0
Lowest Birth Defect NOEL	10.0	3.125	4,000	8.0
ADI	0.025	0.1	0.011	0.01
1-in-a-Million Cancer Probability[b]	0.00004	0.0076	0.000052	0.00021
	Per milligram per kilogram per day over a lifetime			
Cancer Potency	0.025	0.057[c]	0.019[d]	0.0047

[a] Highest dose tested; actual number would be higher.

[b] Lifetime daily dose that would result in a 1-in-a-million chance of person getting cancer from this chemical alone.

[c] Carbaryl itself is considered to be noncarcinogenic; the potency value is for N-nitrosocarbaryl, while the 1-in-a-million probability figure is for the amount of carbaryl required to form enough N-nitrosocarbaryl to pose a 1-in-a-million chance of cancer. As discussed in footnote 1 on page H-14, the cancer potency of N-nitrosocarbaryl could range from 0.057 to 3.6. If the higher potency value were used, the 1-in-a-million probability figure would be 0.00012 mg/kg/day.

[d] Diflubenzuron itself is considered to be noncarcinogenic; statistic is for 4-chloroaniline, which can be produced when diflubenzuron breaks down.

for humans is 0.025. This means that a person would have to receive 0.00004 milligram of acephate per kilogram of body weight every day throughout his or her life to have a 1-in-a-million chance of getting cancer from that chemical alone. For a 154-pound person, that would be 0.0028 milligram per day.

Carbaryl

Threshold Responses. Carbaryl is also considered to be a moderately toxic insecticide. Its main toxic effect is caused by inhibiting cholinesterase. But these effects are more readily reversible than with either acephate or trichlorfon. In addition to animal studies, its effects on humans have been documented in poisoning incidents, worker exposure studies, and volunteer ingestion studies. The laboratory tests have revealed such symptoms as reduced cholinesterase, swollen kidney cells, and various birth defects in animals exposed during gestation.

The Environmental Protection Agency used a NOEL of 10 milligrams per kilogram per day as the basis for setting carbaryl's ADI. This NOEL is for cholinesterase inhibition in rats. EPA applied a safety factor of 100 to the rat NOEL to set the ADI for carbaryl at 0.1 milligram per kilogram per day. For a 154-pound person, this "safe dose" would be 7 milligrams per day.

Based on the weight of the evidence, EPA concluded that carbaryl will not cause birth defects in humans. Further, there was a study to see if the rate of birth defects was higher in areas where carbaryl had been used to control gypsy moths compared to areas where it had not been used. The study found that there was no difference between the rates of birth defects in the two areas. But because of public concern about birth defects, the analysis for this Environmental Impact Statement used the lowest known birth defect NOEL. That NOEL, 3.125 milligrams per kilogram per day, is lower than the one used by EPA to set the ADI. The birth defects showed up in dogs at doses ranging from 6.25 to 50 milligrams per kilogram per day. Birth defect NOELs for other mammals are much higher than the dog NOEL. The lowest non-dog NOEL for birth defects is from a study that used rabbits; that NOEL is 150 milligrams per kilogram per day.

It may be that the results of carbaryl studies using dogs should not be applied to humans. Those studies suggest that dogs might be much more sensitive than other mammals to this chemical. Because of these doubts, EPA has called for more data about how carbaryl affects dogs.

Nonthreshold Responses. While the evidence is not conclusive, carbaryl seems to have a weak ability to cause mutations but no ability to cause cancer. But there is concern that carbaryl could combine with nitrites to form N-nitrosocarbaryl, a compound that can cause mutations and cancer. Such a chemical reaction can take place only under acidic conditions like those found in the human stomach. The N-nitrosocarbaryl produced would pose a cancer risk only if it could remain in the stomach long enough to cause tumors. It is uncertain that N-nitrosocarbaryl could last that long.

In one study, carbaryl and nitrite were fed directly to rats. No tumors were observed even at doses that caused acute poisoning. Even so, this analysis makes the worst case assumption that carbaryl would be converted to N-nitrosocarbaryl in the human stomach. Based on use of the linear model, the cancer potency of N-nitrosocarbaryl was found to be 0.057. This means that a dose of 0.000018 milligram of N-nitrosocarbaryl per kilogram of body weight every day during a person's lifetime could result in a 1-in-a-million chance of that person getting cancer. To get that much N-nitrosocarbaryl, a 154-pound person would have to take in about 0.52 milligram of carbaryl daily.[1]

Diflubenzuron

Threshold Responses. Diflubenzuron is selective in its toxicity. It causes the outside skeleton of insects to rupture when they molt. It is considered to be only slightly toxic to humans. There is no evidence that diflubenzuron causes birth defects. The main health concern is that diflubenzuron is known to raise the levels of sulfhemoglobin and methemoglobin in blood. This effect could impair the blood stream's ability to carry oxygen. The lowest NOEL, 1.1 milligrams per kilogram per day, is for this type of response. EPA has set the ADI at 0.011 milligram per kilogram per day. For a 154-pound person, this "safe dose" would be 0.77 milligram per day.

Nonthreshold Responses. Laboratory studies indicate that diflubenzuron does not cause mutations or cancer. But there might be some risk of cancer associated with exposure to this chemical because one of its breakdown products--4-chloroaniline--might cause cancer. The evidence about 4-chloroaniline is suggestive, not conclusive. But this analysis makes the worst case assumption that 4-chloroaniline can cause cancer in humans. The cancer potency (calculated in Appendix I) would range from 0.0036 to 0.034. Based on a potency of 0.019, a 154-pound man would have to be exposed to about 0.0036 milligram of 4-chloroaniline per day throughout his life to have a 1-in-a-million chance of getting cancer from this chemical. For diflubenzuron to be the source of this exposure, something like the following would need to occur: Every day of his life, the man would have to eat an entire fish that had been exposed to 0.01 milligram of diflubenzuron.

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[1] As discussed in court during Oregon Environmental Council v. Kunzman, the cancer potency of N-nitrosocarbaryl could range from 0.057 to 3.6 depending on what study is used to calculate the potency. If the higher potency value were used, a 154-pound person would have to ingest 0.0084 milligram of carbaryl every day to have a 1-in-a-million chance of getting cancer. This range of potency values is discussed further in Appendix I.

Trichlorfon

Threshold Responses. Trichlorfon is a moderately toxic insecticide; its main toxic effect is caused by inhibiting cholinesterase. The lowest NOEL is 1.0 milligram per kilogram per day for reduced cholinesterase levels in dogs. At considerably higher doses, lab animals have shown some changes in their immune systems and had birth defects. The immune system NOEL is 20 milligrams per kilogram per day and is from a study that used rats. The lowest birth defect NOEL also is from a rat study. This NOEL is 8 milligrams per kilogram per day. A study using hamsters found no birth defects at doses 25 times higher than that. The World Health Organization has set trichlorfon's ADI at 0.01 milligram per kilogram per day. For a 154-pound person, this "safe dose" would be 0.7 milligram per day.

Nonthreshold Responses. For this analysis, it is assumed that trichlorfon could cause genetic mutations and cancer in humans. While tests using whole animals have been inconclusive, laboratory studies using bacteria, yeast, and mammalian cells indicate that trichlorfon is mutagenic. And while there is no direct evidence that trichlorfon can cause cancer, it is assumed that it can because cancer and mutations form in similar ways. Making this worst case assumption and using the linear model, the cancer potency would be 0.0047. This means that a 154-pound person would have to be exposed to about 0.015 milligram of trichlorfon every day for a lifetime to have a 1-in-a-million chance of getting cancer from this chemical.

EXPOSURE ANALYSIS

For an insecticide to cause harm to a person, two conditions must be met. First, the substance must be in the person's environment. Second, it must enter the body.

Exposure to an insecticide must come from the air that a person breathes, the water the person drinks, or the food the person eats, or the chemical must come into contact with the skin. The amount of chemical in a person's environment is the exposure level.

If the chemical is in the air, it can enter the body through the air passages and lungs (called the inhalation route). If it is on a person's clothes or skin, it must pass through the skin to enter the body (the dermal route). A chemical also could get into the body if the person eats food or drinks water that has insecticide residues (the ingestion, oral, or dietary route). The total amount that actually enters the body is called the dose.

In gypsy moth projects, two groups of people can be exposed. The first group is workers. This group includes supervisors, pilots, truck drivers, mixer/loaders, and observers (including inspectors, scouts, rangers, and ecologists). The second group includes members of the general public living in or near sprayed areas.

To find out how much of the chemical these groups could be exposed to, all likely ways a person could be exposed were identified. Then doses from these exposure routes were estimated using standard methods and assumptions. These doses, along with information from the hazard identification section, are used in the risk evaluation section to assess the health risks to workers and the public from exposure to the insecticides.

Possible Routes of Exposure

To cover most ways a person could be exposed, a set of situations, called "scenarios," is used. These range from situations that possibly could occur during routine spraying operations to an unlikely event, such as accidental spills.

Exposures from Routine Spraying Operations

Workers and the public can be exposed to the insecticides in two ways: directly and indirectly. Direct exposures are when the chemical comes into contact with the skin or a person breathes the spray. (As discussed on page F-34 of Appendix F, inhalation exposures from spraying operations are insignificant.) Thus, observers who happen to be under the spray plane or mixer/loaders who splash the chemical on their hands would get direct exposures. Direct exposure also can come from spray drift.

Indirect exposure comes from touching sprayed things like yard furniture or tools that have residues on them.

Indirect exposure also can come from eating meat or vegetables or drinking water that have insecticide residues. Figures H-4 and H-5 show the possible routes of exposure from routine operations.

Exposures from Accidents

The highest exposures to workers and residents could come from large amounts of insecticide accidentally spilled from an aircraft (an airplane or helicopter) or a truck. If the mixture is spilled onto a person, the primary route of exposure would be through the skin. If it is spilled into a stream or other body of water, the route would be by drinking water or eating fish from that stream. Possible routes of exposure from accidents are shown in Figure H-6.

Estimating Doses

For each chemical in each scenario, a range of doses was obtained. Realistic doses are levels that might realistically occur during routine spraying. Worst case doses are very high estimates of the most a person might get in that scenario. These exposures are based on assumptions that fit the real world (that is, that are plausible) but that always overestimate risk. Certain actions, such as warning people about spraying operations and making sure that spraying takes place only under the right weather conditions, would reduce the likelihood of worst case doses.

Most of the doses in this assessment are based on field studies where carbaryl was used in spraying operations. These studies provide a range of dose levels that actually occurred to both workers and residents during spraying operations that are similar to gypsy moth spraying operations. Because the chemicals are all applied in a similar way and the exposure routes are expected to be similar for all four chemicals, the carbaryl studies are the best source available for estimating doses in this analysis. But there are some uncertainties in using this method because the four chemicals have different properties. For example, the amount of time it takes for the chemicals to break down, or degrade, varies. In addition, the amount of chemical on the skin that will enter into the body (called the dermal absorption rate) varies.

Thus, in extrapolating the doses from the carbaryl studies to the four chemicals discussed here, these differences must be considered. For example, a 10-percent dermal absorption rate is used for all four chemicals even though the estimated absorption rates for each of the chemicals may be lower. In this way, no risks are underestimated. The different degradation rates also are accounted for when

Exposure Scenario	Routes of Exposure
Mixer/loaders	All routes of exposure (dermal, inhalation, and ingestion).
Observers	All routes of exposure.

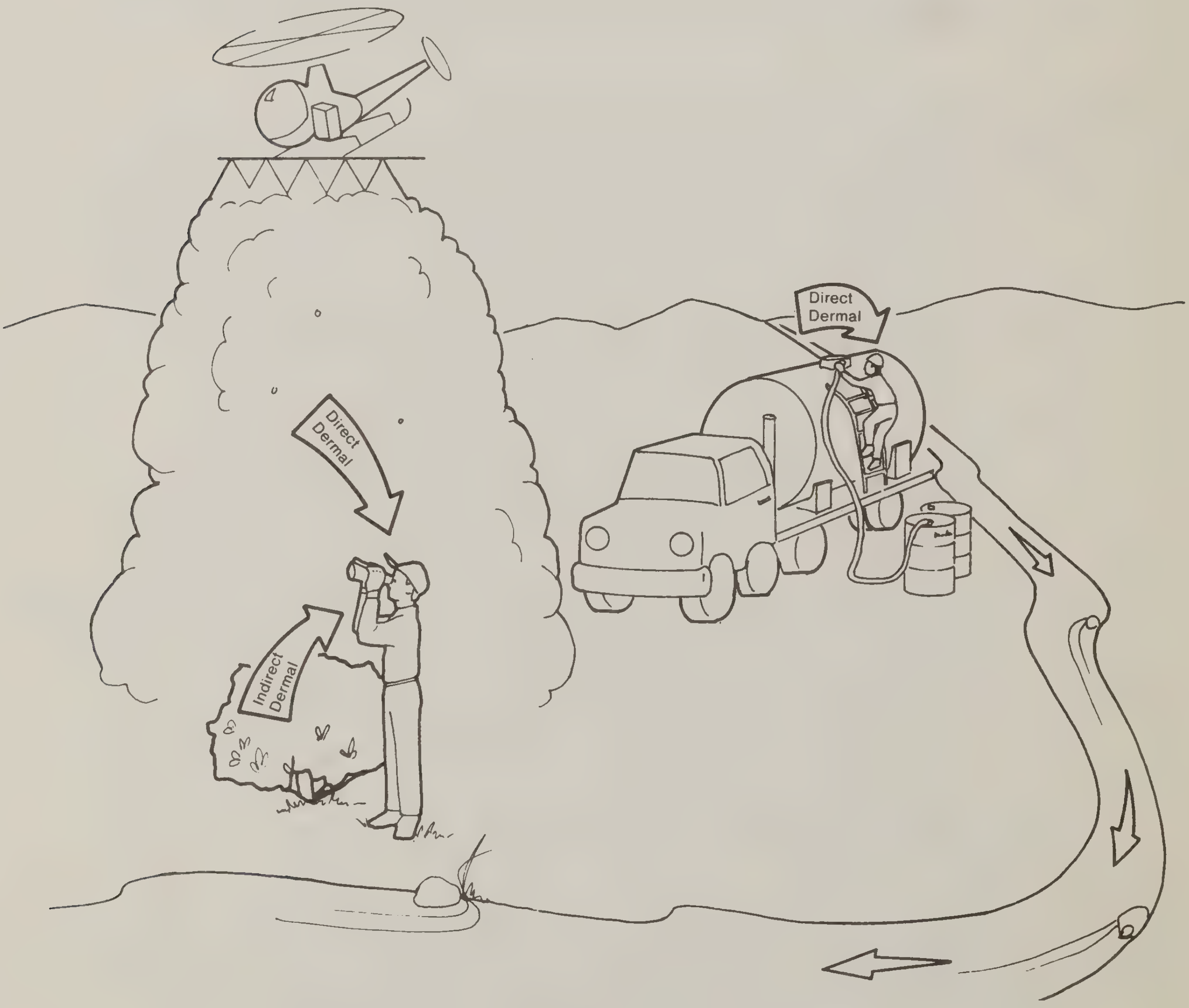
Direct Dermal
Direct Dermal
Indirect Dermal

Figure H-4.--Possible routes of exposure to workers from routine gypsy moth control projects.

Exposure Scenario	Routes of Exposure
Direct	Dermal and inhalation exposure from being outside during a spray operation plus dermal exposure from contact with things like plants, grass, or outdoor furniture.
Indirect	No direct dermal exposure (person is inside during spraying) but indirect exposure from contact with items that have insecticide residues.
Dietary	Ingestion exposure from eating about a pound of fish or meat, a pound of vegetables or fruits, and drinking about a half gallon of water--all of which have insecticide residues.

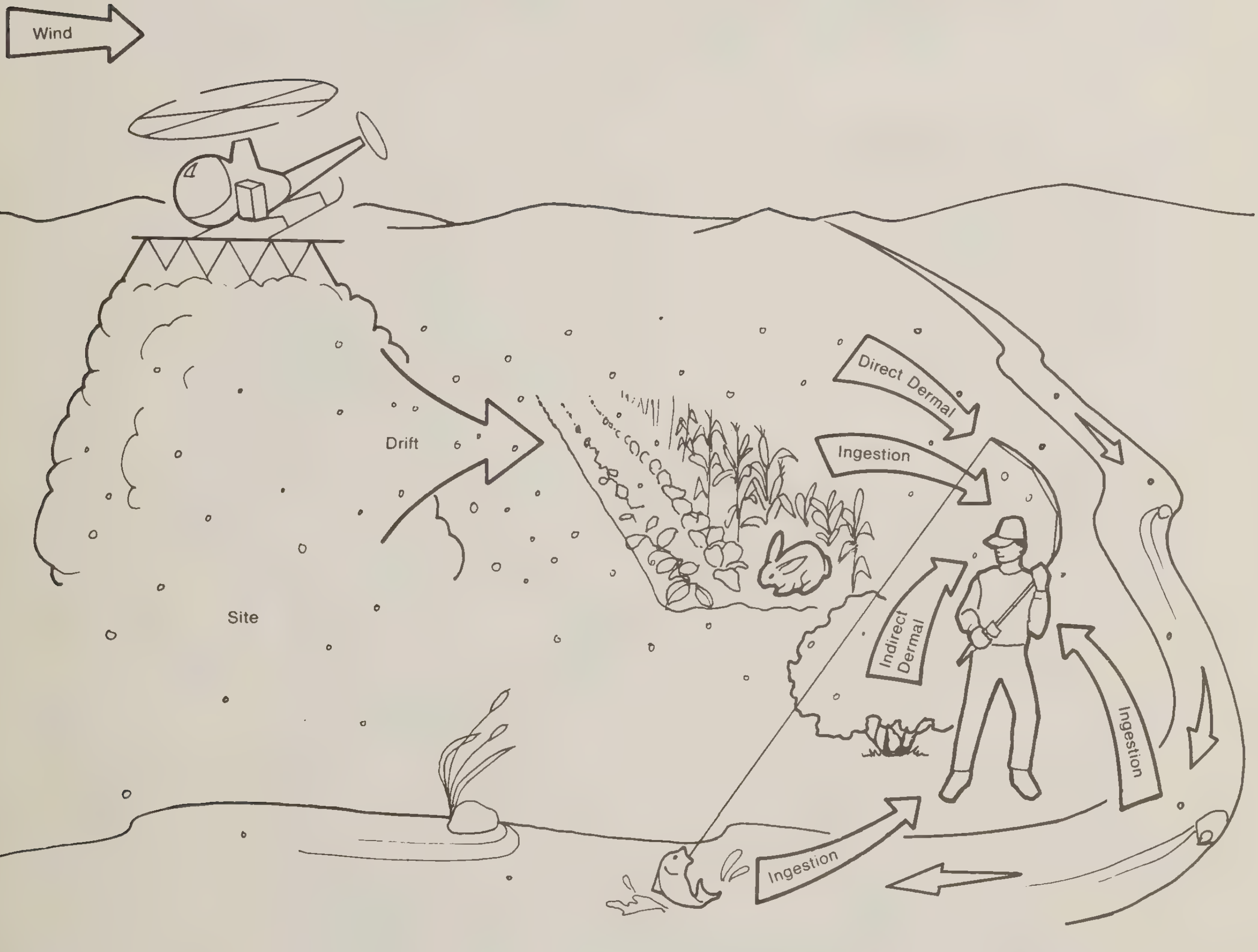
Wind
Drift
Site
Direct Dermal
Ingestion
Indirect Dermal
Ingestion
Ingestion

Figure H-5.--Possible routes of exposure to the general public from routine gypsy moth control projects.

Exposure Scenario	Routes of Exposure
Aircraft Spill	
Partial Dermal	Dermal exposure from the spill of a load of insecticide on a person that enters the body only through those areas not covered by clothes.
Full Dermal	Dermal exposure from aircraft spill that enters the body through exposed skin plus through soaked clothes.
Drinking Water	Ingestion exposure from drinking a half gallon of water from a stream where an aircraft spilled 300 gallons.
Eating Fish	Dietary exposure from eating a pound of fish from the site of a 300-gallon spill.
Truck Spill	
Dermal	Dermal exposure from a truck spill that results in 1 gallon of chemical on the skin.
Drinking Water	Drinking a half gallon of water from a stream that had 2,000 gallons of insecticide mixture spilled into it.
Eating Fish	Eating a pound of fish from a stream in which 2,000 gallons of insecticide had been spilled.

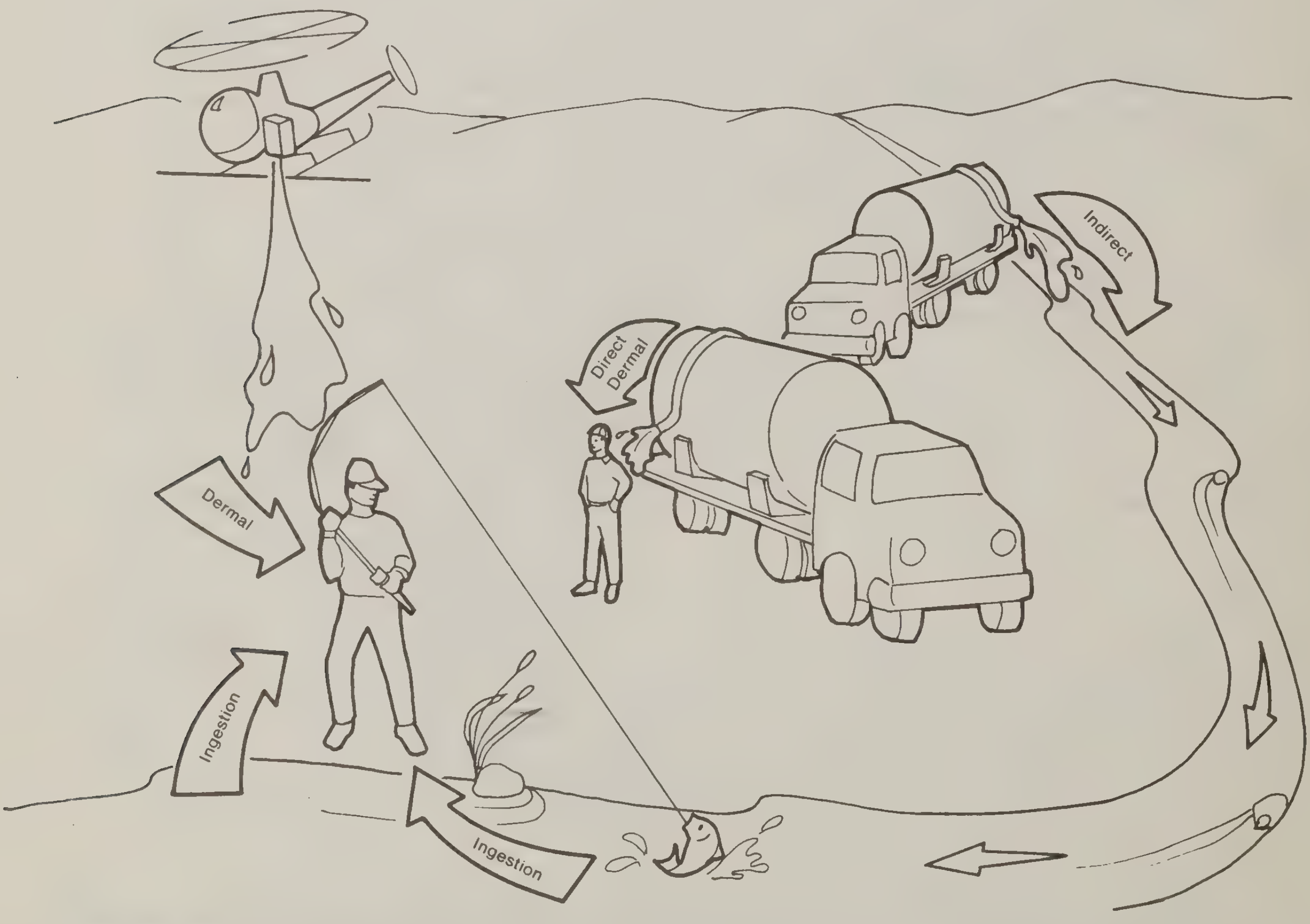

Figure H-6.--Possible routes of exposure from accidents.

determining lifetime doses. The application rate (the amount of active ingredient of chemical applied per acre) is also considered in determining doses. The application rate for both trichlorfon and for carbaryl is 1.0 pound of active ingredient per acre. The application rate for acephate is 0.75 pound per acre, and for diflubenzuron, 0.06 pound per acre.

After the basic dose for each chemical in each scenario was calculated, it was adjusted to account for variations in mixing and application. "Realistic" doses are multiplied by 1.1 to account for normal variations that can increase doses. "Worst case" doses are multiplied by 2.0 to account for abnormal variations that can cause major differences in the amount of spray being deposited. For more detail on mixing and application variations, see pages F-45 through F-47 of Appendix F.

The estimated realistic and worst case doses for all exposure scenarios are shown in Tables H-2 and H-3.

Doses from Routine Operations

Table H-4 summarizes how the realistic and worst case doses were estimated for each scenario for routine operations. (Pages F-27 through F-43 in Appendix F provide specific details about how these doses were determined.) All doses to the public overestimate risk because, in calculating these initial doses, it was assumed that the chemicals do not degrade (that is, their levels do not decline over time) at all. Thus, the doses for each scenario are the highest that could be expected.

The dietary dose is the only dose not based on the carbaryl studies. This dose is calculated based on insecticide residue levels found in meat, fish, vegetables, and water. Realistic and worst case doses from residues in vegetables and fruits were based on studies of residue levels from agricultural applications of these chemicals.

To receive the multiple exposures under the dietary scenario, a person would have to gather and eat the food or drink the water right after the spraying. Although some fruits and vegetables are growing during the spring when gypsy moth spraying occurs, none are mature enough at that time to be harvested. In addition, insecticide residues in vegetables degrade rapidly within 1 or 2 weeks. Thus, the likelihood that people would get doses from eating vegetables or fruit with insecticide residue is very low. But to ensure that even this remote possibility is considered, doses from this food source are included in the dietary dose.

Table H-2.--Estimated realistic doses for exposure scenarios (milligrams per kilogram per day)

Exposure Scenario	Acephate	Carbaryl	Diflubenzuron	Trichlorfon
ROUTINE OPERATIONS				
Workers				
Mixer/loaders	0.035	0.046	0.0028	0.046
Observers	0.0017	0.0022	0.00013	0.0022
General Public				
Direct	0.0017	0.0022	0.00013	0.0022
Drift	0.0011	0.0014	0.000087	0.0014
Indirect	0.00033	0.00044	0.000026	0.00044
Direct plus Dietary	0.025	0.012	0.00072	0.012
Indirect plus Dietary	0.024	0.0098	0.00061	0.0098
Observer plus Dietary	0.026	0.012	0.00072	0.012
Dietary only	0.024	0.0094	0.00059	0.0094
ACCIDENTS				
Aircraft spill				
Dermal (partial)	60	120	2.3	60
Dermal (full)	120	260	3.5	120
Drinking water	0.033	0.077	0.0015	0.033
Eating fish			0.00014[a]	
Truck spill				
Dermal	11,000	27,000[b]	430	11,000[b]
Drinking water	0.25	0.60	0.01	0.23
Eating fish			0.00091[a]	

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[a] Dose is from 4-chloroaniline, a breakdown product of diflubenzuron

[b] The variation factor is not applied. These chemicals are premixed and there is no possibility for mixing error.

Table H-3.--Estimated worst case doses for exposure scenarios
(milligrams per kilogram per day)

Exposure Scenario	Acephate	Carbaryl	Diflubenzuron	Trichlorfon
ROUTINE OPERATIONS				
Workers				
Mixer/loaders	0.15	0.20	0.012	0.2
Observers	0.044	0.058	0.0035	0.058
General Public				
Direct	0.0075	0.010	0.0006	0.010
Drift	0.0049	0.0054	0.00032	0.0054
Indirect	0.0014	0.0018	0.00011	0.0018
Direct plus Dietary	0.11	0.12	0.0079	0.13
Indirect plus Dietary	0.11	0.12	0.0074	0.12
Observer plus Dietary	0.15	0.17	0.01	0.17
Dietary only	0.10	0.12	0.0073	0.12
ACCIDENTS				
Aircraft spill				
Dermal (partial)	110	230	4.2	110
Dermal (full)	220	470	6.4	220
Drinking water	0.060	0.140	0.0028	0.06
Eating fish			0.00025[a]	
Truck spill				
Dermal	19,000	27,000[b]	780	10,000[b]
Drinking water	0.45	0.60	0.018	0.23
Eating fish			0.0017[a]	

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[a] Dose is from 4-chloroaniline, a breakdown product of diflubenzuron.

[b] The variation factor is not applied. These chemicals are premixed and there is no possibility for mixing error.

Table H-4.--Methods for estimating doses from routine operations

Scenario	Realistic Dose	Worst Case Dose
Workers		
Mixer/loader	Based on urine levels in mixer/loaders from carbaryl studies. High range (not average) used.	Based on highest dose level (from urine) from carbaryl studies, as well as studies on other pesticides.
Observer	Based on dose level (from urine) in observers in carbaryl studies.	Assumes observer sprayed directly on 2 square feet of exposed skin.
General Public		
Direct	Based on average exposure levels for residents found in carbaryl studies.	Based on highest exposure level to residents reported in the carbaryl studies.
Drift	Dermal dose based on drift found offsite in a number of relevant studies; two-thirds of the amount deposited onsite gets offsite under routine assumptions.	Dermal dose based on two-thirds of amount deposited onsite under worst case assumptions (for example, application rate is multiplied by 2).
Indirect	Based on lowest dose found for observers in carbaryl studies.	Based on highest dose found for observers in carbaryl studies
Dietary	The estimated dietary exposure level is the sum of the following: eating a pound of meat (from two sources), fish, and vegetables and drinking a half gallon of water--all of which have insecticide residues.	
	Goat/Rabbit--Goats and rabbits are exposed through both dermal and inhalation routes.	Goats and rabbits are exposed to very high levels of insecticide.
	Fish--Based on residue levels found in actual field test of water intentionally directly sprayed with carbaryl.	Based on direct application to stream 6 inches deep with no dilution.
	Vegetables--Based on the low range of residue data from agricultural applications assuming vegetables are picked and eaten the same day as treatment.	Based on high range of residue data from agriculture applications.
	Water--Concentration in water calculated by the same method as for fish residues.	

To determine the highest cumulative doses a person could get from all sources, some doses were combined. For example, the dietary dose was added to the direct, indirect, and observer doses to show the most the general public could be exposed to during routine operations.

Doses from Accidents

If an aircraft or truck spilled insecticide, workers and the general public could be exposed to much more than they would under routine circumstances. However, such accidents are rare. (The probability of such accidents occurring, which are based on State and Forest Service records of such events, is discussed in more detail in the risk evaluation section.) The assumptions and methods used to determine accidental doses are discussed on pages F-52 through F-55 of Appendix F.

Aircraft Spills. For aircraft spills, doses are calculated for dermal exposure and for drinking water and eating fish that have insecticide residues. The load dumped is assumed to be 300 gallons. This is the size of the load typically used in gypsy moth spraying. This load could spill over land or into water. Exposures are based on the assumptions that the spill over land hit a person or the person drank water or ate fish containing the spilled chemical.

Truck Spills. For spills from trucks, doses were calculated for dermal exposures and for drinking water or eating fish containing insecticide residue. Because no studies are available on worker exposure from a truck spill, basic dermal doses were based on the assumption that a mixer/loader is exposed to 1 gallon of diluted insecticide in a day. The basic dose from drinking water containing insecticide residue was calculated in the same way as for the aircraft spill. Doses from eating fish (exposure to 4-chloroaniline) are a portion of the amount of diflubenzuron in the fish.

Lifetime Exposures and Doses

To determine the risks of long-term health effects such as cancer from exposure to the four chemicals, it is necessary to know how much of the chemical a person might get in a lifetime. For the linear cancer model (described in the hazard identification section), the total lifetime dose must be expressed in terms of average daily dose. The average lifetime daily realistic and worst case doses are summarized in Table H-5.

Routine Operations

To find the lifetime doses from routine spraying, the following information is needed:

- The length of the lifetime (assumed to be 70 years)
- The type and number of gypsy moth projects that could take place in the same area during a lifetime
- The number of days the insecticides might be sprayed during each project
- The amount of chemical a person could be exposed to during each day

Table H-5.--Average lifetime daily doses for realistic and worst case exposures from eradication and suppression projects (milligrams per kilogram per day)

	Eradication Projects (6 exposures)		Suppression Projects (10 exposures)		Combined Eradication and Suppression Projects (16 exposures)[a]	
Exposure Scenario	Realistic	Worst Case	Realistic	Worst Case	Realistic	Worst Case
Acephate						
Direct plus Dietary	0.000058	0.00018	0.000097	0.00031	0.00016	0.00049
Observer plus Dietary	0.000058	0.00027	0.000097	0.00045	0.00016	0.00072
Carbaryl (N-nitrosocarbaryl)						
Dietary	0.000000036	0.00000045	0.000000059	0.00000074	0.000000095	0.00000119
Diflubenzuron (4-chloroaniline)						
Dietary (eating fish)	0.000000068	0.0000014	0.00000011	0.0000023	0.000000079	0.0000037
Trichlorfon						
Direct plus Dietary	0.000017	0.00020	0.000028	0.00033	0.000045	0.00053
Observer plus Dietary	0.000017	0.00021	0.000028	0.00035	0.000045	0.00056

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[a] See pages H-27 and H-28 and I-23 to I-25 for a discussion of cumulative exposures from combined eradication and suppression projects.

- The length of time the insecticide stays in meat, on vegetables, or in water (called persistence)

Persistence is considered only in determining lifetime doses (not in determining initial doses). Lifetime doses are based on all the doses a person could get during the time it takes for the chemical to degrade.

The two types of spraying projects generally used to control gypsy moths are eradication and suppression projects. Eradication projects are used in areas where the gypsy moth has become established by artificial means. For example, a mobile home can carry the insect into a new area where it becomes established. Suppression projects usually are conducted only in areas where the gypsy moth is established and spreads naturally.

For eradication projects, it is assumed that the area may be sprayed as many as three times over a 6-week period and that the gypsy moth could be artificially introduced into the same area twice during 70 years. Thus, in a lifetime, a person living in the same place could be exposed to insecticide six times from eradication projects.

It is assumed that the chemical is sprayed only once in each suppression project and that such a project could be conducted in the same area every 7 years. Thus, a person would be exposed 10 times over 70 years from suppression projects.

Because the gypsy moth spreads naturally and can become established in new areas, suppression projects could take place in areas that also had eradication projects. If so, a person could get as many as 16 exposures in a lifetime (6 for eradication projects and 10 for suppression projects). A detailed discussion of how average lifetime doses are determined is on pages F-73 through F-86 of Appendix F.

Carbaryl (N-nitrosocarbaryl). Carbaryl does not cause cancer. However, there is some concern that nitrites and carbaryl might react to produce N-nitrosocarbaryl, which can cause cancer. Because the probability of this taking place is unknown, it was assumed that it does. In humans, this reaction could occur only in the stomach, so the only dose considered is the dietary dose.

The total dietary dose of carbaryl is the sum of the doses that a person could get during the time the chemical remains, or persists, in food sources (meat and vegetables) or water. Carbaryl residues drop to zero in 7 days in meat, in 14 days in vegetables, and in 4 days in water. To ensure that the total dose was not understated, the analysis used the longest period (14 days).

The total doses of carbaryl are translated into N-nitroso-carbaryl doses. To get the average lifetime dose, the total dose over the 14 days is multiplied by 6, 10, or 16 (the number of exposures in a lifetime) and then divided by the number of days in a lifetime (25,550). These doses are shown in Table H-5.

Acephate and Trichlorfon. For acephate and trichlorfon, the average lifetime doses were calculated for the two highest combined exposures. The first includes those living in the treatment area during spraying and eating food and drinking water having residues (the direct plus dietary exposure scenario). The second includes an initial direct exposure during spraying, as well as other exposures from residues in food and water (the observer plus dietary exposure scenario).

The average lifetime doses for acephate and trichlorfon are based on the first dose from the spraying and the secondary exposures to residues in food and water. For acephate, 20 days is required in both the realistic and worst case for residues to degrade. For realistic doses of trichlorfon, it takes 2 weeks for the residues to degrade completely.

For worst case doses, it takes 60 days for the chemical to degrade. To get the average lifetime doses from acephate and trichlorfon (shown in Table H-5), the total dose over the degradation period (20 days, 14 days, or 60 days) is then multiplied by the number of exposures in a lifetime (6, 10, or 16) and then divided by the number of days in a lifetime. (Calculations are shown in detail on pages F-76 through F-80 of Appendix F.)

Diflubenzuron (4-chloroaniline). Studies show that diflubenzuron does not cause cancer. But it is not known for sure whether a breakdown product of diflubenzuron, 4-chloroaniline, can cause cancer. Because of this uncertainty, it is assumed that 4-chloroaniline can cause cancer. The risk of cancer would come from eating meat or fish, where 4-chloroaniline can be concentrated. Because fish would have the highest level of residue, realistic and worst case doses from eating fish were calculated.

The doses of 4-chloroaniline are figured as a percentage of the estimated doses of diflubenzuron. The residues of 4-chloroaniline in fish would degrade to zero in 60 days. The doses over the 60-day period are then multiplied by the number of exposures in a lifetime and then divided by the number of days in a lifetime to get the average lifetime realistic and worst case doses from 4-chloroaniline. (Page I-21 in Appendix I shows these calculations in more detail.)

Accidents

To evaluate cancer risk from accidental exposures to the chemicals, average lifetime doses from these exposures must be determined. (These are discussed in detail on pages F-84 through F-86 of Appendix F.) Dermal doses are multiplied by the dermal absorption rate (10 percent) and then divided by days in a lifetime to get the average lifetime dose from accidents. Oral doses are divided by days in a lifetime.

It should be noted that averaging a single large dose over 70 years creates uncertainty in the cancer risk calculations discussed in the risk evaluation section that follows. That is, the actual risks of getting cancer could be higher or lower than those presented in this analysis.

For example, a single large dose from an accident, which might occur only once in a lifetime, might overwhelm the body's normal ability to get rid of the poison or repair the damage it caused. In that case, the risk of getting cancer would be higher than the risk determined here.

On the other hand, the risk might be lower than the risk stated here. For humans, the chemical would be in the body for only 1 day in a 70-year lifetime. To get the cancer potency, animals were given daily doses over a period about as long as the animal's natural lifetime.

Population at Risk

For every acre sprayed, it is estimated that 14 persons from the general public could be exposed during gypsy moth operations. (A detailed discussion of how this number was determined is on pages F-64 and F-65 in Appendix F.)

Forest Service records show that, on average, the insecticides have been used yearly on about 385,000 acres. Assuming 14 people per acre, about 5.4 million potentially could be affected by gypsy moth control projects.

Among these 5.4 million, some individuals or groups (for example, infants) might be more sensitive than most people to the four insecticides. It is not possible to determine how many people would fall into the "sensitive category." But specific potential health effects on this group are discussed in the risk evaluation section. To be cautious, the NOELs for this group were reduced by an arbitrary safety factor of 100; that is, they are 100 times lower than those used for the general public.

RISK EVALUATION

What, then, are the health risks to workers and the general public from exposure to these insecticides?

To determine these risks, the estimated exposure levels (from the exposure analysis and shown in Tables H-2 and H-3) are compared with the toxic effect levels (from the hazard identification and shown in Table H-1). This comparison indicates whether harm would be caused if the exposure occurs. But the odds of these exposures occurring is another question.

Some exposure scenarios are much less likely to occur than others. Because of the 10-percent correction for normal variations in mixing and spraying, even the realistic doses in the routine scenarios are higher than should occur in most sprayings. The worst case doses in the routine scenarios are far more unlikely. At most, there would be only 1 worst case exposure for every 500 realistic exposures.

A review of accidents in insecticide-spraying projects suggests that there would be 1 aircraft spill on land for about every 2,000 flights and 1 spill on water for about every 17,000 flights. Based on the yearly number of flights in the past, this suggests that gypsy moth projects could have about 2 aircraft spills every 3 years. Spills would involve worst case loads once every 800 years.

While truck accidents could lead to the highest exposures, these exposures are the least likely to occur. Based on national accident statistics for similar types of vehicles, trucks used in gypsy moth projects would have 1 accident for every 3 million miles traveled. Truck accidents involving spills would occur less than once every 8 million miles. Assuming that trucks carrying insecticides travel an average of 100 miles per project, truck spills on land would occur once every 93,000 trips and on water once every 800,000 trips. The odds of a truck spill on land occurring in association with a worst case dose is about 1 in 50 million; on water, 1 in 460 million. (To see how these odds were calculated, see pages F-55 to F-62 in Appendix F.)

Threshold Responses

Comparisons of threshold responses for all four chemicals under all exposure scenarios are listed in Table H-6. When the estimated dose might occur more than once, it is compared to the acceptable daily intake. High one-time doses from accidental spills are compared to the acute lethal dose for dermal exposure. (If the reader wants specific information about how many times the dose is above or below the ADI, NOEL, or LD_{50} values, refer to Tables 8 through 15 on pages F-123 through F-130 of Appendix F.)

Table H-6.--Comparison of estimated doses to established acceptable daily intakes and acute lethal doses for each insecticide under different exposure scenarios

	Realistic Exposures		Worst Case Exposures	
	Relationship of Estimated Dose to:		Relationship of Estimated Dose to:	
Exposure Scenario	Acceptable Daily Intake (ADI)	Acute Lethal Dose (Dermal LD_{50})	Acceptable Daily Intake (ADI)	Acute Lethal Dose (Dermal LD_{50})
	Acephate			
Routine Operations				
Workers				
Mixer/Loaders	Above		Above	
Observers	Below		Above	
General Public				
Direct	Below		Below	
Drift	Below		Below	
Indirect	Below		Below	
Direct plus dietary	Same		Above	
Indirect plus dietary	Same		Above	
Observer plus dietary	Same		Above	
Dietary only	Same		Above	
Accidents				
Aircraft spill				
Dermal (partial)		Below		Below
Dermal (full)		Below		Below
Water drinking	Same		Above	
Truck spill				
Dermal		Same		Above
Drinking water	Above		Above	
	Carbaryl			
Routine Operations				
Workers				
Mixer/Loaders	Below		Above	
Observers	Below		Below	
General Public				
Direct	Below		Below	
Drift	Below		Below	
Indirect	Below		Below	
Direct plus dietary	Below		Above	
Indirect plus dietary	Below		Above	
Observer plus dietary	Below		Above	
Dietary only	Below		Above	
Accidents				
Aircraft spill				
Dermal (partial)		Below		Below
Dermal (full)		Below		Below
Water Drinking	Same		Same	
Truck spill				
Dermal		Above		Above
Drinking water	Above		Above	

Table H-6. (Continued)--Comparison of estimated doses to established acceptable daily intakes and acute lethal doses for each insecticide under different exposure scenarios

	Realistic Exposures		Worst Case Exposures	
	Relationship of Estimated Dose to:		Relationship of Estimated Dose to:	
Exposure Scenario	Acceptable Daily Intake (ADI)	Acute Lethal Dose (Dermal LD_{50})	Acceptable Daily Intake (ADI)	Acute Lethal Dose (Dermal LD_{50})
	Diflubenzuron			
Routine Operations				
Workers				
Mixer/Loaders	Below		Same	
Observers	Below		Below	
General Public				
Direct	Below		Below	
Drift	Below		Below	
Indirect	Below		Below	
Direct plus dietary	Below		Same	
Indirect plus dietary	Below		Same	
Observer plus dietary	Below		Same	
Dietary only	Below		Same	
Accidents				
Aircraft spill				
Dermal (partial)		Below		Below
Dermal (full)		Below		Below
Drinking water	Below		Below	
Truck spill				
Dermal		Below		Below
Drinking water	Same		Above	
	Trichlorfon			
Routine Operations				
Workers				
Mixer/Loaders	Above		Above	
Observers	Below		Above	
General Public				
Direct	Below		Same	
Drift	Below		Below	
Indirect	Below		Below	
Direct plus dietary	Same		Above	
Indirect plus dietary	Same		Above	
Observer plus dietary	Same		Above	
Dietary only	Same		Above	
Accidents				
Aircraft spill				
Dermal (partial)		Below		Below
Dermal (full)		Below		Below
Water Drinking	Above		Above	
Truck spill				
Dermal		Above		Above
Drinking water	Above		Above	

Note: The estimated doses are shown in Tables H-2 and H-3; the ADIs and LD_{50}s are shown in Table H-1.

It must be emphasized that the comparisons with ADIs and LD_{50}s could be misleading. The estimated doses from spraying mostly would be one-time or of short duration. Yet the ADIs are doses that can be safely taken every day for a lifetime. Because of the safety factors used to set ADIs, it may be possible that doses just above the ADI would not cause harm. For example, if a safety factor of 100 is used to set an ADI, what is the significance of a dose that exceeds the ADI for a single day but still provides a margin of safety of 99? In any case, all doses that exceed the ADI are examined to determine potential health effects. With regard to the dermal LD_{50}s, it is important to remember that, for all four chemicals, these were the highest doses tested and that they were not lethal. So a dose that exceeds the dermal LD_{50} might not be as harmful as it seems.

Except for most doses resulting from accidents, all realistic doses to the general public are the same as or below the ADIs. In most cases, workers also receive realistic doses that are below the ADIs. Mixer/loaders could receive realistic doses of acephate and trichlorfon that are slightly above the ADIs.

For both the general public and workers, some routine exposures under the worst case lead to doses above the ADIs. All such exposures to the general public include eating food and drinking water containing spray residues.

Every dose from routine operations would be below the level that could cause birth defects if you accept EPA's conclusion that carbaryl does not cause birth defects in humans. (The issue of carbaryl's ability to cause birth defects is discussed further on pages H-13 and H-36.)

Estimated doses at or below the ADI are considered safe for the general public and are unlikely to pose any health risks. Estimated doses above the ADI or near or above the LD_{50} are another matter. In those cases it is necessary to take a closer look to determine the following:

- How close are the doses to the NOELs? That is, what are the margins of safety? (Figure H-7 illustrates the concept of margin of safety.)
- What might happen to the exposed person?
- What are the odds that the exposure would occur?

Even if an estimated dose is below the ADI, it still might affect sensitive individuals. The risk analysis assumes that sensitive individuals are 100 times more sensitive to

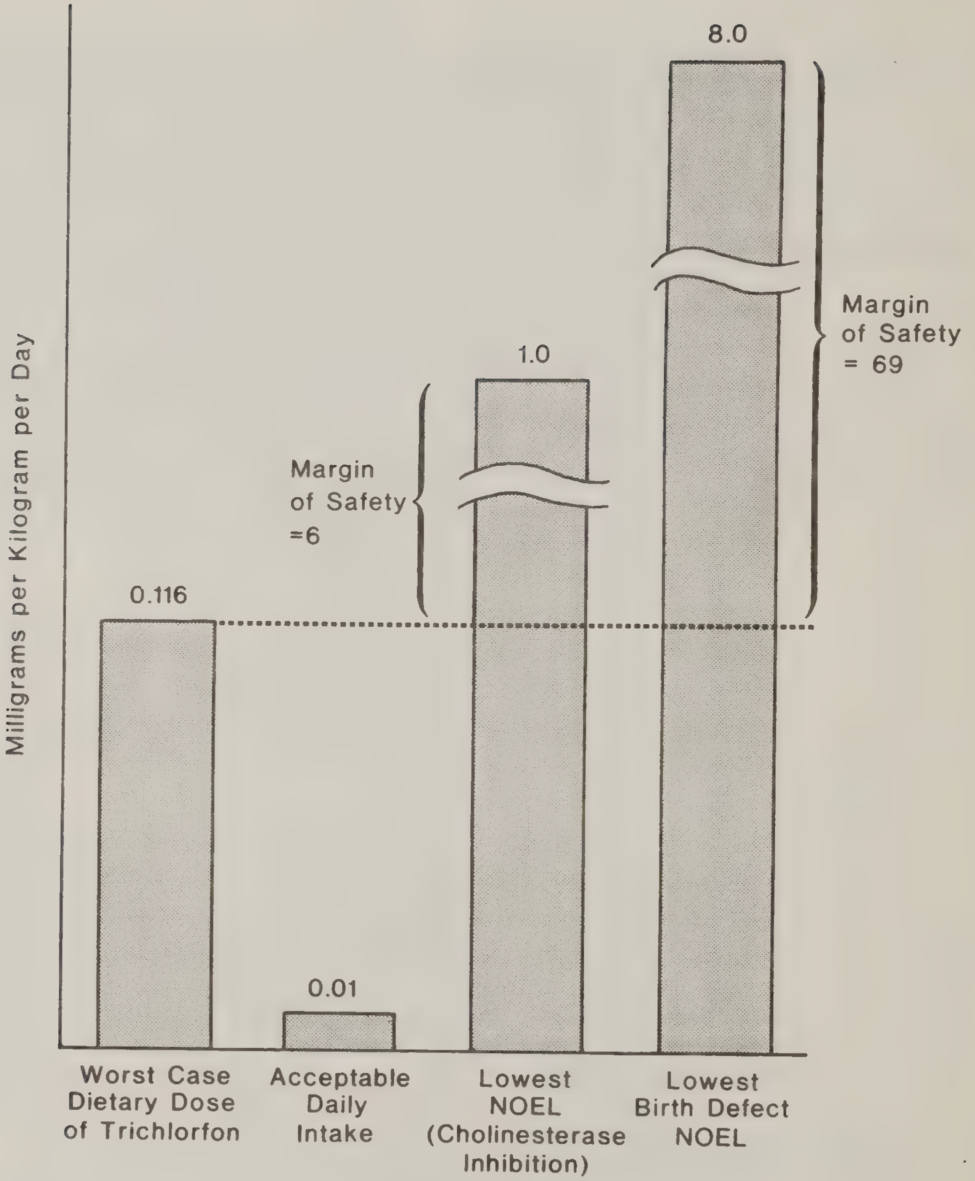

Figure H-7.--How margins of safety are determined. These margins are found by comparing the estimated doses (shown in Tables H-2 and H-3) with the NOELs (shown in Table H-1). This example compares the estimated worst case dietary dose of trichlorfon with two of that chemical's NOELs. The estimated dose is 6 times below (one-sixth) the lowest NOEL and 69 times below the lowest birth defect NOEL.

chemicals than the general population. So it is also necessary to look closely at these possible effects.

The following sections provide these closer looks.

Acephate

Routine Operations--Workers. The highest dose is the worst case dose to mixer/loaders. This dose is 6 times above the

ADI and half the lowest NOEL, which is for cholinesterase inhibition. Realistic doses to mixer/loaders are also slightly above the ADI and only 7 times below the lowest NOEL. Thus, these workers might have temporary symptoms of cholinesterase inhibition. These symptoms could include headaches, dizziness, blurred vision, or nausea. But it should be noted that a study of plant and lab workers exposed to acephate found no effects on blood cholinesterase levels.

Routine Operations--General Public. Worst case doses that include the dietary component are 4 to 6 times above the ADI. These worst case doses are half the lowest NOEL, so there might be some of the same symptoms of mild cholinesterase inhibition. Again, these effects would be temporary. The chance of birth defects is remote since such effects have not been induced in lab animals at the highest doses tested.

Routine Operations--Sensitive Individuals. All doses from routine operations could lead to cholinesterase inhibition in sensitive individuals. Realistic doses could result in headaches or dizziness. Worst case doses could result in more severe symptoms, such as nausea and convulsions.

Accidents. Dermal exposures from aircraft spills range from 47 to 177 times below the dermal LD_{50}. While there should be no deaths, exposed persons could suffer from headaches, dizziness, nausea, abdominal cramps, diarrhea, or sweating.

Dermal exposures from a tank truck accident are up to twice as high as the dermal LD_{50}. The effects of such an exposure are not known for sure, though cholinesterase inhibition surely would occur. Possible symptoms include unconsciousness, breathing problems, convulsions, vomiting, harm to the nervous system, or even death if the exposed person does not get prompt medical treatment. This type of poisoning can be treated with atropine sulfate, a common antidote. While the truck spill poses the greatest health hazard, it has the lowest odds of occurring--about 1 in 100,000.

The worst case dose from drinking water contaminated by an aircraft spill is twice the ADI (one-fourth the NOEL). The realistic dose from drinking water with insecticide from a truck spill is 10 times above the ADI and the same as the lowest NOEL. The worst case drinking water dose from a truck spill is twice the NOEL. These three doses may result in temporary signs of cholinesterase inhibition (such as headaches, dizziness, and nausea).

Carbaryl

Routine Operations--Workers. The worst case dose to mixer/loaders is twice the ADI, but 16 times below the lowest NOEL. The lowest NOEL used in Appendix F is for birth defects in dogs. But there is doubt that this NOEL can be related to humans. As discussed in the hazard section, dogs seem uniquely sensitive to carbaryl. Birth defect NOELs from tests using other mammals are much higher, and EPA has concluded that carbaryl will not cause birth defects in humans. Other than tests that used dogs, the lowest NOEL for cholinesterase inhibition is from a study using rats and is 10 milligrams per kilogram per day. The margin of safety with respect to this rat NOEL is 50. So mixer/loaders might have some mild ill effects of cholinesterase inhibition.

Routine Operations--General Public. It is unlikely that there would be any ill effects to the general public from realistic doses resulting from carbaryl spraying. The four worst case doses that include food and water containing spray residues are slightly above the ADI. An observer who eats food and drinks water containing spray residues receives a dose 18 times below the lowest (dog) NOEL. So, assuming that carbaryl can cause birth defects in humans, the worst case doses might pose some risk of birth defects to the general population. Using the lowest non-dog NOEL for birth defects, these worst case doses would have a margin of safety of about 1,000. This margin of safety, along with the low probability of a worst case exposure occurring (less than 1 chance in 500), suggests that the risk of birth defects is quite low. This same worst case dose (observer plus dietary) has a margin of safety of 57 compared to the rat NOEL for cholinesterase inhibition. Thus, an observer who eats food and drinks water containing spray residues might have mild adverse effects.

Routine Operations--Sensitive Individuals. For sensitive individuals, realistic doses of carbaryl pose a threat of causing birth defects if the individual responds in the same way as dogs--that is, if the dog NOEL is used for determining the margin of safety. If the lowest non-dog NOEL (reduced by a factor of 100) is used for comparison, the margin of safety would be 125. Therefore, no birth defects would be expected as a result of these realistic doses. But the worst case doses could be seen as causing birth defects in sensitive individuals even when the doses are compared to non-dog NOELs. All doses to sensitive individuals could lead to kidney problems and symptoms of cholinesterase inhibition.

Accidents. All dermal exposures received from aircraft spills are far below the dermal LD_{50}. Realistic and worst case dermal exposures received from a truck spill are three times above the dermal LD_{50}. Without prompt medical help, such a dose could cause convulsions, shortness of breath, unconsciousness, or even death. Again, there is a common antidote that could reverse most symptoms. There is about 1 chance in 100,000 that such a spill would occur.

Drinking water containing insecticide from a truck spill results in a dose that is 6 times above the ADI. This dose is 5 times below the lowest NOEL and could result in cholinesterase inhibition. If the water were drunk for a week or two, there might be a chance of birth defects. There is a 1 in a million chance that such a spill would occur.

Diflubenzuron

Routine Operations--Workers. All doses are below or equal to the ADI.

Routine Operations--General Public. All doses are below or equal to the ADI.

Routine Operations--Sensitive Individuals. There may be two groups that would be at greater risk than the public at large. They are people with genetic defects that make them prone to having methemoglobin in the blood, and very young infants who lack enzymes that can reduce the level of methemoglobin. Worst case doses that include the dietary component are roughly the same as the lowest NOEL reduced for sensitive individuals. To avoid potential ill effects in these two groups, efforts should be made to keep them from being exposed.

Accidents. All dermal exposures result in doses that are below the dermal LD_{50}. All drinking water doses are below or equal to the ADI.

Trichlorfon

Routine Operations--Workers. Realistic doses to mixer/loaders are 5 times above the ADI. These doses are 22 times below the lowest NOEL, which is for cholinesterase inhibition. Worst case doses to observers are 6 times above the ADI (17 times below the lowest NOEL), and worst case doses to mixer/loaders are 20 times above the ADI (5 times below the NOEL). These workers thus run the risk of cholinesterase inhibition. Symptoms, which would be temporary, could range from eye irritation to headaches and nausea.

Routine Operations--General Public. The only doses that might be harmful to the general public are the worst case doses that include food and water containing spray residues. These doses range from 12 to 17 times above the ADI. The margin of safety with respect to the lowest NOEL ranges from 6 to 8. Symptoms might include eye irritation, dizziness, or headaches. These same worst case doses could cause birth defects based on the rat NOEL of 8 milligrams per kilogram per day. But this is uncertain since the rat NOEL was the highest dose tested. The next highest birth defect NOEL is from a study that used hamsters and is 25 times higher. If that NOEL were used, the margin of safety would be more than 1,000.

Routine Operations--Sensitive Individuals. All doses from routine operations could cause cholinesterase inhibition in sensitive individuals. The worst case doses that include the dietary components would exceed the reduced NOELs by a factor of 10 or more. Symptoms could range from headaches to convulsions. Based on the rat birth defect NOEL, all doses could cause birth defects in sensitive individuals. If the hamster NOEL were used, just the worst case doses that include the dietary component could be seen as leading to birth defects in sensitive individuals.

Accidents. Truck accidents could be quite hazardous. Dermal exposures from truck accidents are 5 times the dermal LD_{50}. The poisoning could affect the lungs and nervous system, and it could cause convulsions, unconsciousness, or even death. Again, there is a common antidote, so most symptoms could be reversed with prompt medical help.

The doses received from drinking water containing trichlorfon residues from accidental aircraft spills range from 3 to 6 times above the ADI (30 to 17 times below the lowest NOEL). Doses from drinking water following a truck spill are 23 times above the ADI (one-fourth the NOEL) for both the realistic and worst cases. These drinking water doses could lead to mild symptoms of cholinesterase inhibition. These temporary symptoms could include eye irritation, dizziness, headaches, or nausea.

Nonthreshold Responses

Cancer

It was assumed that using any of the four chemicals might lead to cancer. The probability of cancer being caused by exposure to a chemical is determined by multiplying its cancer potency for humans (as derived through use of the linear model) by the lifetime average daily dose under each of the exposure scenarios.

The cancer probabilities are listed in Table H-7. The numbers in this table are weighted lifetime risks for all

Table H-7.--Weighted cancer risk to individual if exposed to insecticide under different exposure scenarios (chances in a million over a lifetime)

Exposure Scenario	Acephate	Carbaryl[a]	Diflubenzuron[b]	Trichlorfon
Routine Operations				
Eradication projects	1.4	0.0021	0.0012	0.08
Suppression projects	2.4	0.0035	0.0023	0.13
Cumulative eradication and suppression projects	3.8	0.0055	0.0035	0.21
Accidents				
Aircraft Spill				
Dermal (partial)	5.7			1.1
Dermal (full)	12			2.2
Drinking water	0.032	0.0004		0.0068
Eating fish			0.0001	
Truck spill				
Dermal	1,000			190
Drinking water	0.24	0.0031		0.042
Eating fish			0.00067	

[a] Statistic is for risk of cancer from N-nitrosocarbaryl. If the higher cancer potency discussed on page H-14 were used, all the cancer risks would still be less than 1 in a million.

[b] Statistic is for risk of cancer from 4-chloroaniline.

exposed people. They assume a person is exposed to the chemical, but they also take into account the odds of realistic and worst case exposures occurring. (At most, there would be 1 worst case dose for every 500 realistic doses.) A worst case dose could raise the odds of cancer two- to twelve-fold.

The meaning of the numbers in Table H-7 can be lost without reference to other commonly known health risks. For example, the riskiest of the routine scenarios in terms of cancer is the one in which acephate is used to both eradicate and suppress gypsy moths. Under that scenario, the odds of getting cancer from acephate are estimated to be about 4 in a million. A person would have the same chance of getting cancer by smoking about 8 cigarettes in his or her lifetime.

To evaluate fully the risk of cancer from an accident, the chances of the accident occurring also should be considered. For example, the odds are about 2 in a million that an individual completely doused with trichlorfon from an airplane spill would get cancer. At the same time, such a spill only would occur in about 1 of every 2,000 trips. Even then it would be highly unlikely for a person to be doused. Thus, when the cancer risks related to accidents are considered along with the low probability of accidents occurring, the chances of cancer resulting from an accident become remote.

Acephate. The cancer risk was calculated for persons who receive a direct application of acephate and who eat and drink food and water containing spray residues. These are the people in the general public who receive the highest exposures.

The weighted lifetime cancer risk from exposure to acephate is 1.4 in a million for eradication projects, 2.4 in a million for suppression projects, and 3.8 in a million for the combination of both. There would be less than one incidence of cancer for every 300,000 acres sprayed. In the past, acephate has been used on less than 1,000 acres per year. Thus the added risk of cancer from using acephate could be about 0.003 incidence per year in the exposed population of 14,000 people.

Carbaryl. For carbaryl, the only doses related to cancer are those obtained through eating and drinking. This is because N-nitrosocarbaryl could be formed only through ingestion of carbaryl.

The weighted cancer risk for carbaryl (from the formation of N-nitrosocarbaryl) is 2.1 in a billion for eradication projects, 3.5 in a billion for suppression projects, and about 5.5 in a billion for the combination of both. (If the higher cancer potency discussed on page H-14 were used, the cancer risks would still be less than 1 in a million.) There would be less than one incidence of cancer for every 150 million acres sprayed. In the past, carbaryl has been used on about 82,000 acres per year. Thus the added risk of cancer from using carbaryl could be about 0.0004 incidence per year in the exposed population of 1.14 million people.

Diflubenzuron. The potential cancer risk from diflubenzuron comes from eating meat or fish containing 4-chloroaniline, a breakdown product of diflubenzuron. The data in Table H-7 assume that a person eats meat or fish exposed to diflubenzuron.

The weighted cancer risk from eating fish or meat containing 4-chloroaniline (from the breakdown of diflubenzuron) is about 1.2 in a billion for eradication projects, 2.3 in a billion for suppression projects, and 3.5 in a billion for the combination of both. There would be less than one incidence of cancer for every 300 million acres sprayed.

In the past, diflubenzuron has been applied to about 141,000 acres per year. Thus the added risk of cancer from using diflubenzuron could be about 0.0005 incidence per year in the exposed population of 2 million people.

Trichlorfon. The cancer risk from exposure to trichlorfon was calculated for persons who receive a direct application and who then eat and drink food and water containing spray residues. Those with lower exposures will have lower cancer risks.

The weighted cancer risk from exposure to trichlorfon in eradication projects is less than 1 in 10 million. The weighted lifetime cancer risk for exposure from suppression projects is about 1 in 7 million, with the weighted lifetime risk from combined projects being about 2 in 10 million. There would be less than one incidence of cancer for every 5 million acres sprayed. Each year, approximately 161,000 acres have been sprayed with trichlorfon. Thus the added risk of cancer from using trichlorfon could be about 0.03 incidence in the exposed population of 2.25 million people.

Heritable Mutations

The risk of heritable mutations is based on the overall evidence of whether or not the chemicals are mutagenic in humans. As indicated in the hazard identification, diflubenzuron is considered to be non-mutagenic; acephate probably cannot cause mutations in whole mammals; carbaryl is only weakly mutagenic; while trichlorfon appears to be a mutagen that can reach germ cells. At the worst case, the probability of acephate, carbaryl, or trichlorfon causing mutations would be no greater than the probability of it causing cancer.

Synergistic and Cumulative Effects

Because of chemicals already in the environment, it is possible that the risks from using these four insecticides might be greater than described.

First, any of these insecticides might combine with different chemicals in the environment. By doing so, they might create effects that are greater than the sum of their separate effects. This process is called synergism. Since many possible combinations could occur, the effects of synergism are hard to predict. But based on studies of carbaryl and other chemicals, a 10-fold increase in toxic levels in isolated instances seems to be the most that could happen. This would be the worst case. Most margins of safety would still be acceptable for the general public, but sensitive individuals could be at risk.

Second, any of the four chemicals might be in the environment from other sources. So gypsy moth spraying could add to amounts that are already there. Especially in the case of carbaryl or acephate, homeowners might be using these chemicals in their gardens. Other sources might be food and spray drift from farm areas where these chemicals are used. But data on chemical residues in food suggest that there would be little, if any, cumulative effect from these four insecticides. (For more details about synergistic and cumulative effects, see pages F-101 to F-104 in Appendix F.)

PREPARERS

This plain language summary of the health risk analysis was prepared under contract by Labat-Anderson Incorporated. The principal preparers were the following:

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Appendix I
Clarification of Information about the Toxicity of Acephate, Carbaryl, Diflubenzuron, and Trichlorfon

INTRODUCTION

This appendix corrects, clarifies, or explains information in the 1985 Final Environmental Impact Statement, including Appendix F. Most of this information was presented in court during Oregon Environmental Council v. Kunzman.

REVIEW OF TOXICITY STUDIES

This section describes more completely the toxicity information that was summarized in Tables 1 through 7 in Appendix F. This information is being included to clarify for the reader the potential hazards of the insecticides. In addition, this information clarifies the background and basis for selecting the no-observed-effect levels (NOELs) used in the worst case analysis. This section also provides the descriptive background needed to identify possible health effects resulting from exposure to insecticides used to control the gypsy moth.

Acephate

Acute Toxicity

Based on an acute oral LD_{50} (median lethal dose) in rats ranging from 866 mg/kg (females) to 945 mg/kg (males), acephate can be classified as a moderately toxic insecticide (USEPA, 1984a). An acute delayed neurotoxicity study did not produce leg paralysis in rats exposed to 375 mg/kg/day, which was the highest dose tested (USEPA, 1982). The acute dermal LD_{50} for rabbits was reported to be greater than 10,000 mg/kg (USEPA, 1984a).

Studies examining the toxicity of acephate in birds (*Junco hyemalis*), cotton plants, insects, and fish have detected residue levels of the insecticide methamidophos (Zinkl et al., 1981; Geen et al., 1981; and Bull, 1979). The metabolite methamidophos is produced as a result of the deacetylation of acephate. Because methamidophos is considerably more toxic than acephate, with a severely toxic LD_{50} of 29.9 mg/kg (Thomson, 1979), there is concern about the fate of methamidophos in nontarget organisms. Methamidophos has been reported to be rapidly produced and eliminated in fish and insects (Geen et al., 1981). Residues of methamidophos were primarily found in insects. The authors of a study conducted to examine the toxicity of acephate on aquatic organisms have concluded that the effect of acephate would be transitory and localized (Geen et al., 1981).

Subchronic Toxicity

The predominant toxic effect seen in test subjects after exposure to acephate is a decreased level of red blood cell, plasma, and brain cholinesterase. An *in vitro* cholinesterase inhibition assay using rat, monkey, and human cells resulted in poor inhibition of acetyl cholinesterase and cholinesterase activities in all test species (USEPA, 1984a). A 21-day rat feeding study resulted in a NOEL less than 30 ppm (1.5 mg/kg/day); 30 ppm was the lowest dose tested and resulted in a 21-percent inhibition of red blood cell cholinesterase during the second test week and a 15-percent inhibition during the third test week (USEPA, 1984a). A 21-day dermal rabbit study resulted in 51 to 54 percent inhibition of red blood cell cholinesterase at dose levels of 0.5, 1.0, and 2.0 g/kg (USEPA, 1984a). A 33- to 34-day oral cholinesterase study during which monkeys were exposed to acephate resulted in a 50-percent reduction of plasma, red blood cell, and brain cholinesterase levels, and a 5- to 17-percent reduction in the level of cerebrospinal cholinesterase (USEPA, 1984a). A 90-day rat cholinesterase study established a NOEL of 5 ppm (0.25 mg/kg) for brain, red blood cell, and plasma cholinesterase levels (USEPA, 1984a).

Chronic Toxicity and Oncogenicity

A 2-year validated Industrial Bio-Test dog feeding study established a systemic NOEL greater than 100 ppm (2.5 mg/kg/day) based on the absence of toxic systemic effects; however, a cholinesterase NOEL of 30 ppm (0.75 mg/kg/day) was reported with reduced levels of red blood cell, plasma, and brain cholinesterase observed at 100 ppm (2.5 mg/kg/day) (USEPA, 1984a).

A 28-month oncogenic rat feeding study resulted in the establishment of a NOEL at 5 ppm (0.25 mg/kg/day) based on inhibition of plasma, red blood cell, and brain cholinesterase levels at the higher doses tested. Histopathological examination of tissue specimens revealed no evidence of carcinogenic effects at the highest dose level, which was 700 ppm (105 mg/kg/day) (USEPA, 1982).

A 2-year oncogenic study during which mice were exposed to 1,000 ppm (150 mg/kg/day) of acephate resulted in a 15.8 percent incidence of liver tumors and a 19.7 percent incidence of excessive noncancerous cell growth in females. At this dose level, weight alteration was observed in the livers, kidneys, brain, and ovaries, and decreased body weight gain was also observed. Liver and lung injuries were observed at all testing levels (USEPA, 1984a).

Teratogenicity

Teratogenic effects have not been induced in laboratory animals upon maternal exposure to acephate during gestation. A validated Industrial Bio-Test rat teratology study reported a teratogenic NOEL greater than 200 mg/kg (highest dose tested) (USEPA, 1984a; USEPA, 1982). A rabbit teratology study also resulted in a teratogenic NOEL greater than the highest dose level of 10 mg/kg; however, a maternal toxic NOEL of 3 mg/kg was reported based on increased nasal discharge and increased incidence of abortion (USEPA, 1984a).

Mutagenicity

Acephate has produced weakly mutagenic results in studies with bacteria, fungal cultures, and nonactivated mammalian cells in culture (USEPA, 1984a). Negative results were reported for bacterial DNA repair assays, activated mammalian cells in culture, the mouse dominant lethal test, the sister chromatid exchange assay and chromosome aberration tests in mice and monkeys (USEPA, 1984a). Acephate was positive upon exposure to a yeast DNA repair assay and a sister chromatid exchange assay using Chinese hamster ovary cells (USEPA, 1984a).

Carbaryl

A summary of established NOELs for carbaryl for various mammals is shown in Table I-1. This table clarifies Table 2 of Appendix F (pages F-115 and F-116).

Acute and Subchronic Toxicity

Based on the acute oral LD_{50} for rats of 270 mg/kg, carbaryl can be classified as a moderately toxic insecticide (USEPA, 1984b). The acute oral LD_{50} for dogs was reported to be less than 500 mg/kg; for monkeys it was found to be greater than 1,000 mg/kg (USEPA, 1984b). The acute dermal LD_{50} for rats was reported to be greater than 4,000 mg/kg and to be greater than 5,000 mg/kg for rabbits (USEPA, 1984b).

Acute and subchronic human exposure to carbaryl has been documented in poisoning reports, worker exposure studies, and volunteer ingestion studies. Ingestion of 2.8 mg/kg of carbaryl (Sevin formulation) resulted in epigastric pain and sweating. These effects were relieved by the administration of the antidote atropine sulfate (Harry, 1977). Ingestion of carbaryl at dose levels of 0.25, 0.5, 1.0, and 2.0 mg/kg by 10 volunteer subjects resulted in nausea and vomiting in one test subject at the highest dose tested, Table I-1.--Summary of established no-observed-effect levels (NOELs) for carbaryl for various mammals

Test Animal	Type of Test	Results	Reference
		General Toxicity	
Rat	2-year feeding	Systemic NOEL = 200 ppm LOAEL = 400 ppm (swelling of kidneys and liver)	Carpenter et al., 1961
	7-day feeding	ChE NOEL = 10 mg/kg/day LOAEL = 50 mg/kg/day (53% decrease in ChE)	U.S. EPA, 1984b
Dog	1-year feeding	ChE NOEL > 7.2 mg/kg/day	Carpenter, 1961
		Systemic NOEL = 1.8 mg/kg/day LOAEL = 7.2 mg/kg/day (swelling in kidney)	Carpenter, 1961
Human	Subacute	NOEL = 0.06 mg/kg/day LOAEL = 0.13 mg/kg/day (reversible decrease of reabsorption of amino acids by proximal tubules)	U.S. EPA, 1984b
Pig	Neurotoxicity	LOAEL = 150 mg/kg/day	Smalley et al., 1969

Table I-1 (continued).--Summary of established no-observed-effect levels (NOELs) for carbaryl for various mammals

Teratogenic/reproductive			
Mouse	Teratogenic (diet)	Teratogenic NOEL > 1,166 mg/kg/day Maternal LOAEL = 1,166 mg/kg/day (decreased weight gain) Fetotoxic LOAEL = 1,166 mg/kg/day (reduced embryo weight)	Murray et al., 1979
	(gavage)	Teratogenic NOEL > 150 mg/kg/day Maternal LOAEL = 150 mg/kg/day (decreased weight gain, ChE inhibition)	Murray et al., 1979
Rabbit	Teratogenic (gavage)	Teratogenic NOEL = 150 mg/kg/day Teratogenic LOAEL = 200 mg/kg/day (omphalocele) Maternal NOEL = 150 mg/kg/day (weight loss)	Murray et al., 1979
	Teratogenic (diet/capsule)	Teratogenic NOEL > 200 mg/kg/day	Robens et al., 1969
Rat	Teratogenic (diet)	Teratogenic NOEL > 500 mg/kg/day Maternal LOAEL = 500 mg/kg/day (decreased weight gain) Fetotoxic LOAEL = 500 mg/kg/day	Weil et al., 1972
	Reproductive (3 generation) (diet)	Reproductive NOEL > 10 mg/kg/day Maternal LOAEL = 10 mg/kg/day Fetoxicity LOAEL = 10 mg/kg/day (dilated uterine gland in third generation pups)	Weil et al., 1972
	Reproductive (3 generation) (diet)	Reproductive NOEL = 200 mg/kg/day	Weil et al., 1973

Table I-1 (continued).--Summary of established no-observed-effect levels (NOELs) for carbaryl for various mammals

	gavage	Reproductive NOEL = 25 mg/kg/day Maternal LOAEL = 100 mg/kg/day (decreased weight gain, ChE inhibition) Fetotoxic LOAEL = 100 mg/kg/day (decreased number of fetus)	Weil et al., 1973
Guinea Pig	Teratogenic (diet)	Teratogenic NOEL > 300 (HDT) Maternal NOEL > 300 Fetotoxic NOEL > 300	Weil et al., 1973
		Teratogenic NOEL < 300	Robens et al, 1969
	Teratogenic (gavage)	Teratogenic NOEL > 200 (HDT) Maternal LOAEL = 200 mg/kg/day (decreased weight gain) Fetotoxic NOEL > 200 mg/kg/day (HDT)	Weil et al., 1973
Dog	Teratogenic (diet)	Teratogenic NOEL = 3.125 Teratogenic LOAEL = 6.25 (lack of tail, abdominal fissure, failure of skeletal formation and extra toes)	Smalley et al., 1978
		Mutagenicity	
Mouse	Dominant lethal	NOEL > 200 mg/kg/day	Epstein et al., 1972

but no toxic effects were observed at the other dose levels (Harry, 1977). Subacute dermal and inhalation exposure of carbaryl production workers at 0.44 to 4.9 mg/m^3 did not produce abnormal sperm count or infertility after a 1-year exposure period (USEPA, 1984b).

Subchronic and acute exposure to carbaryl has resulted in decreased cholinesterase levels and decreased reabsorption by proximal tubules of the kidney in test animals and humans. Cholinesterase is instrumental in the normal propagation of nerve impulses because of its role in bond cleavage of the neurotransmitter acetylcholine. Both these toxic effects are transitory and have not resulted in permanent physiological damage to the exposed individuals (Harry, 1977, and Wills et al., 1968).

An acute oral cholinesterase LD_{50} rat study reported significant depression of plasma, red blood cell, and brain cholinesterase in the surviving test animals. A second acute oral cholinesterase study resulted in depression of the red blood cell cholinesterase level in rats after exposure to carbaryl (in propylene glycol) at the dose of 12.5 mg/kg after 1 and 4 hours (USEPA, 1984b). A 7-day rat cholinesterase study reported a NOEL of 10 mg/kg, with decreased levels of red blood cell cholinesterase at the lowest-observed-adverse-effect level (LOAEL) of 50 mg/kg (USEPA, 1984b). Dermal application of carbaryl (5 percent Sevin 85W) to 10 human test subjects resulted in depressed red blood cell cholinesterase levels after 24 hours; however, 5 days after exposure, the cholinesterase levels had returned to normal (Harry, 1977).

A 6-week subacute human ingestion study during which doses of 0.06 mg/kg and 0.13 mg/kg were administered daily resulted in no toxic effects at the lower dose level. A reversible decrease in the reabsorption capacity in the proximal tubule of the kidney was observed in test subjects after the 6-week exposure to 0.13 mg/kg (Wills et al., 1968).

Chronic Toxicity

A 1-year dog feeding study resulted in morphological changes in the kidneys of test animals but no apparent decrease in cholinesterase levels. The cholinesterase NOEL was reported to be greater than 7.2 mg/kg (highest dose tested) (USEPA, 1984b). A systemic NOEL of 1.8 mg/kg and a LOAEL of 7.2 mg/kg were reported based on diffuse cloudy swelling or vacuolization of kidney cells (USEPA, 1984b). Similar histological effects have been observed in the kidneys of rats and monkeys after exposure to carbaryl (Wills et al., 1968).

A 2-year rat feeding study reported a systemic NOEL of 200 ppm (10 mg/kg/day). At the highest dose level of 400 ppm (20 mg/kg/day), morphological changes characterized by cloudy swelling were observed within tubules of the kidney and hepatic cords of the liver (USEPA, 1984b).

Teratogenicity and Reproduction

A study was conducted to evaluate the teratogenic potential of carbaryl administered by gavage or in the diet to mice and rabbits during days 6 through 15 of gestation. Dietary administration to mice resulted in no teratogenic effects. A teratogenic NOEL for mice greater than 1,166 mg/kg/day (only dose tested) was reported for dietary exposure, and a teratogenic NOEL greater than 150 mg/kg/day (highest dose tested) was reported for exposure by gavage. Fetotoxic effects in mice characterized by decreased maternal weight gain and reduced embryo development were observed at the dietary level of 1,166 mg/kg/day. A maternal NOEL less than 1,166 mg/kg/day was reported based on decreased weight gain. In the gavage study, decreased weight gain and cholinesterase inhibition were reported as maternal toxic effects. Administration of carbaryl by gavage to rabbits resulted in the establishment of a teratogenic NOEL of 150 mg/kg/day based on the occurrence of omphalocele (hernia of the navel). A dose of 200 mg/kg was reported as maternally toxic and 150 mg/kg was reported as mildly maternally toxic when administered by gavage to rabbits (Murray et al., 1979).

A teratology study using guinea pigs, rabbits, and hamsters resulted in teratogenic effects in guinea pigs, but no apparent malformations in hamsters and rabbits. Exposure of hamsters to carbaryl at levels of 125 to 250 mg/kg and rabbits at 50 to 200 mg/kg produced no teratogenic effects. Teratogenic bone defects were observed in guinea pigs at the dose level of 300 mg/kg (Robens, 1969), though another teratology study that exposed guinea pigs to the same dose level produced no teratogenic effects (Weil et al., 1973).

A teratology study that exposed rats to dietary doses up to 500 mg/kg/day of carbaryl did not result in teratogenic effects. Decreased weight gain was reported as a fetal toxic and maternal toxic effect at 500 mg/kg/day (Weil et al., 1972).

A three-generation reproduction study during which rats were exposed daily to carbaryl at 10 mg/kg did not significantly affect fertility, gestation, lactation, or viability of pups (Weil et al., 1972). A second three-generation rat reproduction study established a reproductive NOEL of 200 mg/kg (highest dose tested) when carbaryl was administered as part of the diet (Weil et al., 1973).

A teratology study during which beagle dogs were exposed to 50, 25, 12.5, 6.25, and 3.125 mg/kg of carbaryl throughout the gestation period resulted in a teratogenic NOEL of 3.125 mg/kg. Defects included abdominal fissures, failure of skeletal formation, absence of tail formation, and the presence of extra toes (Smalley et al., 1968).

Mutagenicity

A dominant lethal rat mutation assay indicated carbaryl was nonmutagenic at the exposure level of 200 mg/kg (highest dose tested) (Epstein et al., 1972). However, chromosomal assays resulted in the induction of mitotic effects and chromosomal abberations (USEPA, 1984b). The reproductive effects assessment group of the Environmental Protection Agency has concluded that data from mutagenicity studies indicate that carbaryl does not act as a potent mutagen and can be classified as a weak mutagen (USEPA, 1984b).

Oncogenicity

Despite speculation that carbaryl could combine with nitrite compounds to form a carcinogen under acidic conditions similar to those found in the human stomach, the majority of studies examining the carcinogenic potential of carbaryl have been negative. A preliminary report by the carcinogen assessment group concluded that there was no significant increase in the incidence of tumor induction among treated animals relative to control animals (USEPA, 1984b).

A 2-year oncogenicity rat feeding study was negative for carcinogenic effects at 400 ppm (20 mg/kg/day) (the highest dose tested) (USEPA, 1984b). An oncogenicity mouse study during which carbaryl was either given orally at 464 mg/kg for 5 weeks, fed at 14 ppm (2.1 mg/kg/day), or administered under the skin in a single dose of 100 mg/kg did not induce cancer in test animals (USEPA, 1984b). Another 2-year mouse oncogenicity study was negative at the dietary level of 400 ppm (60 mg/kg/day) (USEPA, 1984b). An intraperitoneal oncogenicity study during which mice were administered carbaryl at a dose level of 60 mg/kg/week produced no oncogenic effects in test animals (USEPA, 1984b). The injection under the skin of 10 mg of carbaryl per week for a 20-week test period was negative for oncogenic effects (USEPA, 1984b). The dermal application of a 57-percent water dilution of carbaryl resulted in no oncogenic effects (USEPA, 1984b).

A 22-month rat feeding study at the dose level of 30 mg/kg (highest dose tested) resulted in the induction of malignant tumors in 4 of 12 surviving test animals (USEPA, 1984b). Oncogenic effects also were observed after the subcutaneous administration of 20 mg of carbaryl to 48 rats;

tumors formed in 2 of 10 surviving test animals. However, no significant increase in tumor incidence in treated groups relative to controls was found by the carcinogen assessment group of the Environmental Protection Agency (USEPA, 1984b).

Viral Enhancement

The subject of viral enhancement was discussed on pages 59 and 60 of the Final Environmental Impact Statement. The following information was presented in court (Oregon Environmental Council v. Kunzman) and updates the discussion in the FEIS.

Interactions between viruses and chemical pesticides have been studied because of concern that these interactions may affect human health. This is of concern because of the suggested link between certain viral diseases and Reye's syndrome. The Maine Bureau of Forestry appointed a health advisory panel to review the data available on Sevin-4-oil specific to viral potentiation; in January 1980, the panel released its findings and recommendations (see Appendix B of USDA, 1981). The panel found that "there was a potential but inconclusive health risk of Sevin-4-oil," on the basis of viral potentiation data available, and recommended that the Maine Bureau of Forestry develop more stringent limitations so that "no uninformed, unconsented human exposure occurs during a forest spray operation." Abrahamsen and Jerkofsky (1981) described the enhancing effect of Sevin-4-oil on the replication of varicella-zoster virus (VZV) in cell culture and suggested possible implications of this interaction with regard to Reye's syndrome. Other more recent studies have raised questions and uncertainty about the Abrahamsen and Jerkofsky findings.

Schmidt (1983) found that carbaryl treatment of host cell cultures delays the early spread of simian varicella viral infections. Schmidt suggested that the apparent effect seen by Abrahamsen and Jerkofsky occurred at a point in the growth cycle at which infectivity had reached maximum levels and was declining in untreated cultures, whereas the cells in the treated cultures, spared from early infection, had finally become infected. Schmidt also suggested that the proposed VZV-enhancing effect of carbaryl and the possible implications to Reye's syndrome might be reevaluated. In a recent study by Brookman et al. (1984), various insecticides, solvents, emulsifiers, and mixtures thereof were evaluated to determine whether any were capable of enhancing the sensitivity of cultured mammalian cells to infection with vesicular stomatitis virus. The investigation was replicated in three independent laboratories. None of the compounds, including the insecticide Sevin, significantly enhanced viral infection. This information, in addition to the study by Schmidt (1983), indicates that carbaryl does not significantly enhance viral infections.

N-nitrosocarbaryl Formation

The subject of N-nitrosocarbaryl formation was discussed on pages 60 and 61 of the Final Environmental Impact Statement. The following information was presented in court (Oregon Environmental Council v. Kunzman) and updates the discussion in the FEIS.

Under acidic conditions similar to those found in the human stomach, carbaryl has been nitrosated in the laboratory to the reaction product N-nitrosocarbaryl (Eisenbrand et al., 1975). Elespuru et al. (1974) found that the combination of sodium nitrite (a food additive) with carbaryl in acid solution results in the formation of nitrosocarbaryl. It is thought that human exposure to nitrosocarbaryl could occur from the reaction of carbaryl residues (in food) with sodium nitrite (in saliva or food) in the acid conditions of the stomach. N-nitrosocarbaryl has been characterized as a mutagen and a carcinogen based on positive laboratory studies (Eisenbrand et al., 1976, and Elespuru and Lijinsky, 1973). An oncogenicity rat study resulted in the induction of malignant tumors at the injection site in 14 of 16 test animals after exposure to a dose level of N-nitrosocarbaryl at 1,000 mg/kg (Eisenbrand et al., 1975). Rats that were administered N-nitrosocarbaryl by gavage developed a high incidence of stomach tumors (invasive squamous carcinomas) (Lijinsky and Taylor, 1976). A rat feeding study also resulted in the formation of stomach tumors (Lijinsky and Schmahl, 1978). N-nitrosocarbaryl appears to be a much less effective inducer of mouse skin tumors than other methylating agents such as nitrosomethylurea. Dermal application of N-nitrosocarbaryl (25 microliters of a 0.04 M solution) to the shaved skin of 20 mice led to the induction of skin tumors at the site of application in 8 of the test animals, but only after repeated dermal applications (twice a week for 50 weeks) to shaved skin. These tumors appeared in 1 of 20 animals at week 60, and in 8 of 20 by week 90 (Lijinsky and Winter, 1981). This indicates that N-nitrosocarbaryl could cause cancer in the stomach or on the skin if it could form in the environment as a result of carbaryl applications. However, the literature shows that N-nitrosocarbaryl only can form under conditions similar to those found in the human stomach--not in the air or on skin surfaces.

A bacterial assay study characterized nitrosocarbaryl as a potent mutagen because of the positive mutagenic response of carbaryl in two bacterial systems (Escherichia coli and Haemophilus influenzae) (Elespuru et al., 1974).

Acute Toxicity

Based on acute oral LD_{50} values greater than 4,640 mg/kg in rats and mice, diflubenzuron can be classified as a slightly toxic insecticide (USEPA, 1984c). The acute dermal LD_{50} for rats was reported to be greater than 10,000 mg/kg, and for rabbits it was greater than 4,640 mg/kg (USEPA, 1984c).

Chronic Toxicity

The major toxic effect observed in test subjects upon exposure to diflubenzuron is the formation of sulfhemoglobin and methemoglobin pigments in the circulatory system. Hemoglobin in its nonoxidized state is essential for the transport of oxygen, whereas the oxidized form, methemoglobin, plays no role in oxygen transport. Investigators have suggested that there is a correlation between increased levels of methemoglobin and increased levels of sulfhemoglobin.

An 80-week mouse feeding study established a NOEL of 1.1 mg/kg/day based on the formation of methemoglobin and sulfhemoglobin in the test animals (USEPA, 1984c). A 104-week rat feeding study resulted in a NOEL of 40 ppm (2 mg/kg/day) with increased levels of methemoglobin and sulfhemoglobin observed in test animals (EPA, 1984c). A lifetime oncogenic mouse feeding study also established a NOEL of 16 ppm (2.4 mg/kg/day) based on increased levels of methemoglobin and sulfhemoglobin (USEPA, 1984c).

Teratogenicity and Reproduction

Teratology studies in rats and mice did not result in teratogenic effects at the levels tested (USEPA, 1984c). Maternal toxicity, fetal toxicity, and teratogenic NOELs were established as being greater than 4,000 mg/kg/day (highest dose tested) for both test species (USEPA, 1984c). A three-generation rat reproduction study resulted in no reproductive toxic effects at 10, 20, 40, and 160 ppm (0.5, 1, 2, and 8 mg/kg/day) (USEPA, 1984c; Uniroyal, 1983).

Mutagenicity

Diflubenzuron was found to be nonmutagenic even at high doses (Quarles et al., 1980; MacGregor et al., 1979; and USEPA, 1984c). Concentrations of 500 mg/kg body weight did not produce a mutagenic response in hamster fetal cells (Quarles et al., 1980). Negative results also were obtained for diflubenzuron in the mouse micronucleus test in vivo, the mouse lymphoma mutation assay, and the bacterial Ames mutation assay (MacGregor et al., 1979).

Oncogenicity

No evidence of oncogenicity was observed in any test animals at doses as high as 1,000 ppm (150 mg/kg/day) in the lifetime oncogenic mouse study (USEPA, 1984c). A second oncogenic study that used rats also produced no oncogenic effects even at 10,000 ppm (500 mg/kg/day) (highest dose tested) (USEPA, 1984c). Although diflubenzuron has not been shown to be carcinogenic, one of its metabolic breakdown products, 4-chloroaniline, has been claimed to be a carcinogen. This possibility is discussed in this appendix in the section on cancer potencies.

Possible Dioxin Contamination

The concern that diflubenzuron may possibly be contaminated with "dioxin" became an issue when a list of 60 pesticides possibly contaminated with dioxin was published in the February 20, 1985, issue of Pesticide & Toxic Chemical News. The list, which was from an internal memo prepared by EPA, included diflubenzuron. After discussion with EPA, USDA was able to determine that the list included any pesticide containing a chlorine on benzene ring. EPA also informed USDA that they did not expect any 2,3,7,8-tetrachlorodibenzo-p-dioxin (the one, of 75 possible dioxin compounds, that people refer to as "dioxin") (USEPA, 1985b). Duphar B.V., the registrant of diflubenzuron, has also tested for the possible presence of 2,3,7,8-tetrachlorodibenzo-p-dioxin or tetrachlorodibenzofurans in technical grade diflubenzuron. They found no contamination using a testing method that had a sensitivity of 0.01 ppm (Shadbolt, 1985). From these discussions, USDA concluded that there was no evidence to indicate that diflubenzuron is contaminated with "dioxin."

Trichlorfon

Acute Toxicity

The lowest reported acute oral LD_{50} value for trichlorfon is 144 mg/kg in rats (Mobay, 1981). However, based on the most commonly reported LD_{50} values ranging from 400 to 650 mg/kg, trichlorfon can be classified as a moderately toxic insecticide (International Agency for Research on Cancer (IARC), 1983). The acute dermal LD_{50} for rats was reported to be greater than 2,000 mg/kg (Mobay, 1981). The acute rat inhalation LC_{50} was reported to be greater than 10,000 mg/kg (Mobay, 1981).

Subchronic and Chronic Toxicity

Feeding studies that lasted 3 months to 2 years resulted in cholinesterase inhibition at doses of 1.25 mg/kg/day in dogs and 2.5 mg/kg/day in rats (Doull et al., 1980). A NOEL of 20 mg/kg/day was reported for rats exposed to trichlorfon based on the modification of immunobiological responses in test animals (Olefir as cited in Zamfir et al., 1975). A NOEL of 30 mg/kg/day was reported for modification of vitamin metabolism in rats (Nijegoro et al. as cited in Zamfir et al., 1975), and a NOEL of 57 mg/kg/day was established based on reduced cytochrome oxidase activity in rats (Jdanovici as cited in Zamfir et al., 1975).

Studies using young dogs established a NOEL of 1 mg/kg/day for reduced acetylcholinesterase levels (Jivogliadova et al. as cited in Zamfir et al., 1975). Decreased levels of acetylcholinesterase in dogs resulted in a higher NOEL of 500 mg/kg in another study (Marsh et al. as cited in Zamfir et al., 1975). A systemic dog study reported a NOEL of 100 mg/kg based on the modification of intestinal fermentic function upon exposure to higher levels of trichlorfon (Gheorghien as cited in Zamfir et al., 1975).

Teratogenicity

A teratogenicity study exposed pregnant rats to a single dose of 80 mg/kg at day 9 or day 13 of pregnancy. Daily doses of 8 mg/kg were administered to another group of pregnant rats during the course of pregnancy. The daily dose of 8 mg/kg did not result in birth defects. However, the high single dose administered at day 13 resulted in general edema (accumulation of fluid in body cavities or tissues), hydrocephaly (abnormal increase in cranial fluid), and meningoencephaly (inflammation of the brain and surrounding membranes). Based on the absence of terata at 8 mg/kg, the authors suggest that trichlorfon does not pose a danger to humans because the teratogenic dose of 80 mg/kg is 2,500 times the dose likely during a 24-hour exposure period (Marston and Varonina, 1976).

A teratology study during which gestating rats, hamsters, and mice were exposed to trichlorfon resulted in teratogenic effects in all test species. Administration of trichlorfon to rats by gavage during days 6 through 15 of gestation at the dose level of 480 mg/kg/day (lowest effect level) resulted in malformed fetuses. Embryotoxicity and terata were observed in hamsters at the dose level of 400 mg/kg/day. Fetotoxicity characterized by low weight was observed in mice at doses of 400 mg/kg/day, and terata reported as the occurrence of cleft palates were reported at 500 mg/kg/day (Staples and Goulding, 1979). A NOEL of 200 mg/kg/day was established for hamsters based on the occurrence of embryotoxic and teratogenic effects at 400 mg/kg/day.

Mutagenicity

Both negative and positive results have been reported from various nonmammalian assays examining the mutagenic potential of trichlorfon in bacteria, fungi, plants, and insects. Findings were negative in four assays using bacteria cells, while three other bacterial assays were reported to be positive for mutagenicity (IARC, 1983). The mutagenic effects observed in bacterial cells were characterized by incorrect substitution of bases in the genetic code. Three mutagenicity assays in fungal systems resulted in the stimulation or induction of mutagenic effects during the process of cell division (mitosis) (IARC, 1983). No mutagenic effects were noted in fruit flies (Drosophila melanogaster) exposed to trichlorfon (IARC, 1983). Chromosome damage was observed in the plant Hordeum vulgare after exposure to trichlorfon (IARC, 1983).

The majority of tests using mammalian cell systems have resulted in positive mutagenic effects. Mutagenic effects were induced in assays examining the mutagenic potential of trichlorfon using hamster cells (CHO), and cells derived from the immune circulatory system and bone marrow of mice (IARC, 1983).

Mutagenicity or the induction of unscheduled DNA synthesis was observed after exposure of two human cell lines (epitheliums and fibroblasts) to trichlorfon (IARC, 1983).

Trichlorfon was nonmutagenic in a dominant lethal mouse assay (Becker and Schoneich, 1980).

Oncogenicity

A 90-week oncogenic study during which rats were exposed to 1.98 to 2.08 g of trichlorfon by oral intubation or 1.1 to 1.6 g by intraperitoneal application reported no statistically significant carcinogenic activity in test animals (Mobay, 1979a). A 73- to 75-week oncogenic study during which mice were exposed to trichlorfon at dose levels of 154 to 157.5 mg by oral intubation, 149.7 mg to 160.8 mg by intraperitoneal application, or 375 mg by dermal treatment resulted in no statistically significant occurrence of benign or malignant tumors (Mobay, 1979b).

CLARIFICATION OF CANCER POTENCIES AND RISKS

This section clarifies and recalculates the cancer potency of N-nitrosocarbaryl, and it clarifies the cancer potency of 4-chloroaniline, a breakdown product of diflubenzuron. This section also clarifies cancer risks.

Carbaryl (N-nitrosocarbaryl) In addition to the Eisenbrand et al. (1976) study analyzed in Appendix F, two other studies could be used to calculate the potency of N-nitrosocarbaryl: Lijinsky and Taylor (1976) and Lijinsky and Schmahl (1978). These studies used Sprague-Dawley rats force fed N-nitrosocarbaryl by the gavage method of dosing. The results of the studies are listed below:

Reference	Total dose	Sex	Days in lifetime	Animals with cancer (percent)
Lijinsky and Taylor (1976)	50 mg	F	840	75
	300 mg	M	700	47
Eisenbrand et al. (1976)	5,000 mg/kg	M	167	29
Lijinsky and Schmahl (1978)	600 mg/kg	M	630	21
	600 mg/kg	F	840	57

As discussed previously in this appendix (in the subsection on N-nitrosocarbaryl formation), all reported incidences of cancer were tumors of the forestomach, the dosing site of the gavage method. The studies by Eisenbrand et al. (1976) and Lijinsky and Schmahl (1978) reported 10 and 0 percent cancer, respectively, in the control. The Lijinsky and Taylor (1976) study had only a colony control (zero incidence of cancer). For this analysis we will assume that α, the spontaneous cancer rate, is zero because the cancers noted in the control by Eisenbrand et al. (1976) were different from those caused by the treatment.

The average daily dose (d) was calculated by dividing the total dose by the average time of exposure or lifetime (whichever was longer). It also was assumed that, over the duration of the studies, the female rats weighed an average of 0.1 kg and the males weighed 0.2 kg.

Reference	Sex	Average daily dose (mg/kg/day)
Lijinsky and Taylor (1976)	F	0.59
	M	2.41
Eisenbrand et al. (1976)	M	29.94
Lijinsky and Schmahl (1978)	M	0.95
	F	0.71

The cancer potency, β, was calculated from the linear model by solving for β.

$$R = \alpha + \beta d$$
$$\beta = (R - \alpha)/d$$
$$\beta = R/d \text{ where } \alpha = 0.0$$

Using the Eisenbrand et al. (1976) data for example:

$$\beta = R/d = 0.29/29.94 \text{ mg/kg/day}$$

$$\beta = 0.0097 \text{ (mg/kg/day)}^{-1}$$

Other cancer potencies are:

Reference	Sex	Average dose (mg/kg/day)	Incidence	β(mg/kg/day)$^{-1}$
Lijinsky and Taylor (1976)	F	0.59	0.75	1.3
	M	2.14	0.47	0.22
Eisenbrand et al. (1976)	M	29.94	0.29	0.010
Lijinsky and Schmahl (1978)	M	0.95	0.21	0.22
	F	0.71	0.57	0.80

Extrapolating the cancer potency from rats to humans was done by multiplying by the 1/3 power of the ratio of adult human (70 kg) to adult rat (0.35 kg) weights. For example, using the Lijinsky and Taylor (1976) data for female rats, the cancer rate for humans was computed as follows:

$$\beta(\text{human}) = 1.3 \text{ (mg/kg/day)}^{-1} \times (70/0.35)^{1/3}$$
$$= 7.6 \text{ (mg/kg/day)}^{-1}$$

Cancer potencies for humans from the three studies are estimated below:

Reference	Sex	βHuman (mg/kg/day)^{1}
Lijinsky and Taylor (1976)	F	7.6
	M	1.2
Eisenbrand et al. (1976)	M	0.06
Lijinsky and Schmahl (1978)	M	1.2
	F	4.7

Therefore, the cancer potency in humans of N-nitrosocarbaryl could range from 0.06 to 7.6 $(mg/kg/day)^{-1}$ depending upon which cancer study was used for the calculation. Using arithmetic average cancer potency values from Lijinsky and Taylor (1976) and Lijinsky and Schmahl (1978), the average cancer potency of N-nitrosocarbaryl would be 3.6 $(mg/kg/day)^{-1}$. The cancer potency calculated from Eisenbrand et al. (1976) was not included in the average because the dose tested caused an abnormally high amount of acute toxicity, thereby lowering the cancer potency.

It is important to remember that N-nitrosocarbaryl poses a cancer risk to humans only when it forms in nature and then persists long enough at the site of attack (stomach) to cause a reaction (cancer). When carbaryl and nitrite were fed directly to rats, no tumors were observed even though doses up to those causing acute toxicity were tested (Lijinsky and Taylor, 1977).

The cancer risk to an individual exposed to realistic or worst case dietary doses of carbaryl from eradication projects is calculated as follows:

$R \text{ (risk)} = \beta d = 3.6\ (mg/kg/day)^{-1} \times d$

For a realistic dose:

$R = 3.6\ (mg/kg/day)^{-1} \times 3.56 \times 10^{-8}\ mg/kg/day$
$= 1.3 \times 10^{-7}$ (or a risk of about 1 in 10 million).

For worst case:

$R = 3.6\ (mg/kg/day)^{-1} \times 4.45 \times 10^{-7}\ mg/kg/day$
$= 1.6 \times 10^{-6}$ (or a risk of about 2 in a million).

Cancer risks to an individual exposed to realistic or worst case dietary doses of carbaryl resulting from suppression projects are 2.1×10^{-7} and 2.7×10^{-6}, respectively.

Diflubenzuron (4-chloroaniline)

In the Final Environmental Impact Statement, the risks of cancer from 4-chloroaniline (resulting from the breakdown of diflubenzuron) were estimated based on secondary reports because the full data from the National Cancer Institute (NCI) study (NCI, 1979) were not available. Since then, the Forest Service has obtained the data and recalculated the risks accordingly.

The NCI conducted 2-year cancer bioassays of 4-chloroaniline in both rats and mice (NCI, 1979). Dietary concentrations of 4-chloroaniline were 0, 250, and 500 ppm for rats, and 0, 2,500, and 5,000 ppm for mice. The only cancerous tumors found that were considered to be related to the 4-chloroaniline treatment were fibromas and sarcomas in the spleen of male rats and hemangiomatous tumors in mice. In both cases, the incidences of these tumors were not significantly greater statistically than those found in untreated control animals. However, the findings were considered suggestive of carcinogenicity because of the rarity of these tumors in the spleens of rats in the colonies maintained at NCI. These cancer incidence data therefore were used to calculate the worst case cancer potency of 4-chloroaniline assuming the incidence rate to be significant.

The incidence of tumors in rats and mice was as follows:

Animal	Dose ppm	 mg/kg/day	Incidence of Cancer Males	 Females	Cancer Potency Males	 Females
Rats	0	0	0.05	--	--	--
	250	12.5	0	--	--	--
	500	25	0.20	--	0.034	--
Mice	0	0	0.1	0	--	--
	2,500	375	0.2	0.06	--	--
	5,000	750	0.28	0.19	0.0036	0.0038

The cancer potency, β, was calculated from the linear cancer model

$$R = \alpha + \beta d$$

For example, the cancer potency of male mice was calculated as follows:

$$\begin{aligned} R &= \alpha + \beta d \\ 0.28 &= 0.1 + \beta\ (750\ \text{mg/kg/day}) \\ &= 0.00024\ (\text{mg/kg/day})^{-1} \end{aligned}$$

To extrapolate the cancer potency in mice to humans, the cancer potency was multiplied by the 1/3 power of the ratio of human (70 kg) to mouse (0.02 kg) weight:

$$\beta(\text{Human}) = (70/0.02)^{1/3} \times 0.00024\ (\text{mg/kg/day})^{-1}$$

$$= 0.0036\ (\text{mg/kg/day})^{-1}$$

The cancer potency of 4-chloroaniline therefore could range from 0.0036 to 0.034 $(\text{mg/kg/day})^{-1}$ depending upon which animal study was used to predict cancer in man. The arithmetic average for males of 0.019 $(\text{mg/kg/day})^{-1}$ was used for this analysis.

Based on the recently completed cancer bioassays of diflubenzuron, the cancer risk from this chemical could be considered to be zero (USEPA, 1985a). However, because of the uncertainty about the carcinogenic potential of 4-chloroaniline, there may be some risk of cancer associated with exposure to diflubenzuron.

The theoretical pathways for metabolic breakdown of diflubenzuron in soil, water, plants, and animals were described in the Diflubenzuron Decision Document (USEPA, 1979). Diflubenzuron breaks down into either 4-chlorophenylurea or 2,6-diflurobenzoic acid. The 4-chlorophenylurea can further break down to 4-chloroaniline, which can then degrade to 4-chloroacetanilide. A review of the literature shows that 4-chloroaniline is rarely found in nature. The major metabolites of diflubenzuron are 4-chlorophenylurea, 2,6-diflurobenzamide, or 2,6-diflurobenzoic acid (see USEPA, 1979, and Nimmo et al., 1984). The principal exceptions were fish and animals, with fish having as high as 60 percent of the total diflubenzuron residue found as 4-chloroaniline (USEPA, 1979). Rapid depletion of the residues in fish was reported in the Diflubenzuron Decision Document, but no data on persistence were given.

Arguably, the cancer bioassays for diflubenzuron also have measured the cancer risk associated with 4-chloroaniline because this metabolite would result from any breakdown. However, if a person consumed large amounts of meat or fish containing diflubenzuron, and therefore 4-chloroaniline residues, then he or she possibly could be exposed to higher levels of 4-chloroaniline than were fed the mice and rats in the cancer bioassays. Therefore, the risk of cancer associated with this possible exposure was analyzed.

The realistic and worst case doses of diflubenzuron resulting from residues in fish were estimated to be 0.00003 mg/kg/day (0.06 x 0.0004 mg/kg/day x 1.1) and 0.0006 mg/kg/day (0.06 x 0.0051 mg/kg/day x 2.0) on page F-44. If 60 percent of diflubenzuron is broken down to form 4-chloroaniline, the 4-chloroaniline doses would be 0.0000096 mg/kg/day (0.00003 mg/kg/day x 0.6 x 127.6/210.7) for the realistic case and 0.0002 mg/kg/day (0.0006 mg/kg/day x 0.6 x 127.6/210.7) for the worst case. (The value 127.6/210.7 is the ratio of molecular weights.) Since no persistence data are available, it was assumed that 4-chloroaniline residue in the fish would degrade to zero within 60 days.

The average dose over the 60-day period therefore would be 0.0000048 mg/kg/day (realistic) or 0.0001 mg/kg/day (worst case). The realistic lifetime dose of 4-chloroaniline resulting from eradication projects is then:

$d = 0.0000048$ mg/kg/day x 60 days/project x 6 projects/lifetime x 1/25,550 days/lifetime

$d = 6.8 \times 10^{-8}$ mg/kg/day

The worst case average lifetime dose of 4-chloroaniline from eradication projects is:

$d = 0.0001$ mg/kg/day x 60 days/project x 6 projects/lifetime x 1/25,550 days/lifetime

$d = 1.4 \times 10^{-6}$ mg/kg/day

The average lifetime doses resulting from suppression projects were calculated by multiplying the eradication doses by 1.67, which yields 1.1×10^{-7} and 2.3×10^{-6} mg/kg/day for the realistic and worst case, respectively.

The cancer risk to an individual exposed to diflubenzuron, and therefore possibly to 4-chloroaniline, is calculated as follows for the realistic case from eradication projects:

$$R = \beta d = 0.019\ (\text{mg/kg/day})^{-1} \times 6.8 \times 10^{-8}\ \text{mg/kg/day} = 1.2 \times 10^{-9}$$

Cancer risks to an individual for other realistic or worst case doses are presented below:

	Eradication		Suppression	
	Lifetime Dose	Lifetime Cancer Risk	Lifetime Dose	Lifetime Cancer Risk
Realistic	6.8×10^{-8}	1.2×10^{-9}	1.1×10^{-7}	2.2×10^{-9}
Worst case	1.4×10^{-6}	2.7×10^{-8}	2.3×10^{-6}	4.5×10^{-8}

The cancer risk from accidental spills of diflubenzuron were based on the assumption that an individual would eat 0.5 kg of fish taken from the stream in which the chemical was spilled. To evaluate the risk of cancer from accidental exposure, the single high dose resulting from dermal exposure, water consumption, or fish consumption needs to be expressed in terms of average lifetime dose.

Lifetime Incidences of Cancer Per Acre

To estimate the number of possible incidences of cancer per acre over a lifetime series of applications, the cancer risk is multiplied by the population at risk (14 individuals/acre based on assumptions stated on pages F-64 and F-65 in Appendix F). This translates to the lifetime incidences of cancer per acre for the lifetime number of applications:

Insecticide/ Exposure Scenario	Incidences of Cancer/Acre/Lifetime	
	Suppression (for 10 applications)	Eradication (for 6 applications)
Carbaryl		
Dietary	2.9×10^{-6}	1.8×10^{-6}
Trichlorfon		
Observer and dietary	1.88×10^{-6}	1.12×10^{-6}
Direct and dietary	1.88×10^{-6}	1.12×10^{-6}
Acephate		
Observer and dietary	3.2×10^{-5}	1.97×10^{-5}
Direct and dietary	3.2×10^{-5}	1.97×10^{-5}
Diflubenzuron		
Eating fish/meat	3.2×10^{-8}	1.7×10^{-8}

In a site-specific environmental assessment, total incidences of cancer in the population can be calculated for a single application by dividing incidences of cancer per acre per lifetime by the number of applications (6 or 10) and multiplying by the total number of acres proposed for treatment. For example, for suppression projects, incidences of cancer are calculated as follows (example for carbaryl: 2.9×10^{-6} applications x number of acres treated):

Carbaryl	
Dietary	No. of acres x 2.9×10^{-7}
Trichlorfon	
Observer and dietary	No. of acres x 1.88×10^{-7}
Direct and dietary	No. of acres x 1.88×10^{-7}
Acephate	
Observer and dietary	No. of acres x 3.2×10^{-6}
Direct and dietary	No. of acres x 3.2×10^{-6}

Diflubenzuron

Eating fish/meat	No. of acres x 3.2×10^{-9}

In other words, there would be less than one incidence of cancer if carbaryl were sprayed on 3 million acres, or if trichlorfon were sprayed on 5 million acres, or if acephate were sprayed on 300,000 acres, or if diflubenzuron were sprayed on 300 million acres.

The risk associated with spraying with carbaryl or acephate is less than one case per 500,000 acres treated. The risk associated with spraying diflubenzuron is about one case per 30 million acres treated. Forest Service records show that from 1981 through 1984, carbaryl was used on an average of 81,812 acres yearly while trichlorfon has been applied to an average of 160,867 acres, diflubenzuron has been applied to an average of 141,000 acres, and acephate has been used on less than 1,000 acres. Using these acreage numbers, the added cancer risk from use of acephate would be 0.003 incidences of cancer ($3.2 \times 10^{-6} \times 1,000$) in the exposed population of 14,000 people (14 people/acre x 1,000 acres). There would be 0.02 incidences of cancer ($2.9 \times 10^{-7} \times 81,812$) in the estimated exposed population of 1.14 million people (14 people/acre x 81,812 acres) living on the 81,812 acres treated with carbaryl. There would be 0.0005 incidences ($3.2 \times 10^{-9} \times 141,000$) of cancer in the estimated population of 2 million people living on the acres treated with diflubenzuron. There would be 0.03 incidences ($1.88 \times 10^{-7} \times 160,867$) of cancer in the estimated exposed population of 2.25 million people (14 people/acre x 160,867 acres) living on the 160,867 acres treated with trichlorfon. For the same time period, the USDA Animal and Plant Health Inspection Service (APHIS) participated in carbaryl treatments on an average of 7,852 acres yearly; diflubenzuron, 10,970 acres yearly; and acephate, 73 acres yearly. If the acres treated under APHIS programs were added to acres treated by the Forest Service, only the total incidences of cancer resulting from exposure to carbaryl would increase; 0.024 to 0.026 incidences.

Cumulative Effects

In eradication treatments where a second application of a chemical insecticide is applied 7 to 10 days after the first, exposure levels after the second application will exceed doses discussed under the realistic case for threshold effects for all exposure scenarios. However, the expected realistic exposure level will be less than 2 times the realistic case dose since some degradation of the first application will have occurred before the second one is applied. Expected doses would exceed the ADI for acephate and trichlorfon when dietary components are included (see

Table I-2.--Weighted risk of cancer in a 70-year lifetime from exposure to acephate, carbaryl, diflubenzuron, or trichlorfon as used in gypsy moth suppression or eradication projects

Insecticide Exposure Scenario	Eradication (6 applications)	Suppression (10 applications)	Eradication & Suppression (16 applications)
Acephate			
Direct and dietary	1.4×10^{-6}	2.4×10^{-6}	3.8×10^{-5}
Observer and dietary	1.4×10^{-6}	2.4×10^{-5}	3.8×10^{-6}
Carbaryl (N-nitrosocarbaryl)			
Dietary	1.3×10^{-7}	2.1×10^{-7}	3.4×10^{-7}
Diflubenzuron (4-chloroaniline)			
Dietary	1.2×10^{-9}	2.3×10^{-9}	3.5×10^{-9}
Trichlorfon			
Direct and dietary	8.0×10^{-8}	1.3×10^{-7}	2.1×10^{-7}
Observer and dietary	8.0×10^{-8}	1.3×10^{-7}	2.1×10^{-7}

Tables 8 and 11 of Appendix F) but would be lower than the ADI for carbaryl and diflubenzuron (Tables 9 and 10 of Appendix F).

The expected worst case exposures under the double application eradication approach likely would exceed the ADI only where dietary components are considered for acephate, carbaryl, and diflubenzuron. Expected worst case exposures for all exposure scenarios--except indirect--involving trichlorfon would equal or exceed the ADI.

Given the natural spread and subsequent establishment of gypsy moths into areas previously uninfested, it is possible that suppression projects could be conducted in areas that previously received eradication treatments. For example, Tennessee, Michigan, and Oregon have isolated infestations that currently are being controlled through eradication projects. If the eradication efforts fail, the area (or State) could be declared generally infested. In such cases, it then would be possible to be exposed to both eradication (6 exposures) and suppression (10 exposures) projects in a lifetime, for a maximum of 16 exposures.

For threshold effects, these additional exposures will not result in any human health effects that have not already been discussed in the risk analysis. However, the cumulative impact of these additional exposures will increase the weighted lifetime risk of cancer and heritable mutations. Weighted cancer risks are shown in Table I-2, which updates Table 16 of Appendix F.

In all cases the weighted lifetime risk of cancer and heritable mutations are the same order of magnitude as those associated with suppression or eradication projects alone (less than 1 in a million).

Accidents

The cancer risks associated with the accident scenarios for trichlorfon, carbaryl, acephate, and diflubenzuron are shown in Table I-3.

CLARIFICATION OF EXPOSURE INFORMATION

This section clarifies Appendix F's discussion of how animals are exposed to the insecticides (see page F-35 and F-36), and the assumptions used to determine oral doses to humans from drinking water containing insecticide residues (see pages F-41 and F-42 of Appendix F).

Animals may be exposed to insecticides as a result of ingesting plant material and water, as well as through grooming. Studies of residues of acephate, carbaryl, and trichlorfon on vegetable crops or grass illustrate that

Table I-3.--Cancer risks for accidents

Scenario	Realistic	Worst Case
Trichlorfon		
Aircraft Spill		
Dermal (partial)	1.08×10^{-6}	1.95×10^{-6}
Dermal (full)	2.23×10^{-6}	4.05×10^{-6}
Drinking water	6.08×10^{-9}	1.11×10^{-8}
Truck Spill:		
Dermal	1.87×10^{-4}	1.87×10^{-4}
Drinking water	4.18×10^{-8}	4.18×10^{-8}
Carbaryl		
Aircraft Spill		
Drinking water	2.5×10^{-8}	4.5×10^{-8}
Truck Spill		
Drinking water	1.9×10^{-7}	1.9×10^{-7}
Acephate		
Aircraft Spill		
Dermal (partial)	5.7×10^{-6}	1.0×10^{-5}
Dermal (full)	1.2×10^{-5}	2.2×10^{-5}
Drinking water	3.2×10^{-8}	5.9×10^{-8}
Truck Spill		
Dermal	1.0×10^{-3}	1.9×10^{-3}
Drinking water	2.4×10^{-7}	4.4×10^{-7}
Diflubenzuron		
Aircraft Spill		
Eating fish	1.0×10^{-10}	1.8×10^{-10}
Truck Spill		
Eating fish	6.7×10^{-10}	1.2×10^{-9}

initial residues of insecticides range from 1 to 100 ppm depending on the insecticide and the type of vegetation (see, for example, Chevron, 1973; Pieper, 1979; USEPA, 1983; Back, 1961; and Kuhr and Dorough, 1976). These residues degrade to nondetectable levels within 10 to 14 days on vegetation--except for grass, which can have detectable residues for up to 28 days (Pieper, 1979). There are few data available on the persistence of diflubenzuron on vegetation, but indications are that it persists for a number of months (Wilcox and Coffey, 1978).

The estimated oral doses that result from a person drinking water that contains insecticide residues are based on the following assumptions:

- The insecticide is applied directly to water (contrary to normal operating procedures)

- Water sources will have a minimum average depth of 6 inches.

- Realistic insecticide concentrations are 50 ppb (0.05 mg/liter) for every 1 lb. a.i. per acre application. Worst case concentrations are 0.707 mg/liter for every 1 pound a.i. per acre application of insecticide.

- Daily consumption of water is 2 liters.

- Water consumed is from a surface spring or stream that had direct application.

- Actual persistence times depend on many environmental factors, but data from gypsy moth projects indicate that residues do not remain in running water for more than 2 to 6 days (see, for example, LOTEL, 1975, and Pieper, 1979). Persistence can be much longer in stagnant water bodies (Gibbs et al., 1984), but these are much less likely sources of drinking water.

- It is possible that after spray application, some insecticide might be dislodged by rain within 10 days (based on half-life data) (FEIS, Table 2), and runoff into potable water. This may result in a brief increase in the concentration of insecticide in water. The transitory nature of these residues and the relatively small contribution of drinking water to human exposure compared to the dermal exposure values already estimated (p. F-32) indicate that runoff is not a significant contribution factor for exposure and is thus not considered in this analysis.

ERRATA

The corrections listed in this section should be made to the main text and to Appendix F of the 1985 Final EIS.

Page 6 - In paragraphs 3 and 4, change references from "EPA 1975" to "USDA, 1981a." The reference USDA (1981a) provides more current information.

Page 11 - In third listed item, delete "(Atherton 1977)" reference. This reference is unnecessary.

Page 18 - In paragraph 5, last sentence, add "(1981)" after "Atkins et al." and change "highly toxic" to "relatively nontoxic." These changes correct the citation. The sentence should now read as follows:

Based on work by Atkins et al. (1981), EPA concluded that Sevin XLR is relatively nontoxic to honeybees exposed to direct application.

Page 22 - In paragraph 2, last line, change reference from "PA Bureau of Forestry 1983" to "PA DER, 1983." This change corrects the citation.

Page 32 - In paragraph 6, line 5, delete reference to "Kondakov 1963." This reference is one of several and is extraneous.

Page 43 - In second line on page, change reference from "Wilcox (1973)" to "LOTEL (1975)." The LOTEL (1975) reference provides updated information.

Page 44 - In paragraph 3, line 7, change reference from "Zinkl et al. 1979" to "Zinkl et al., 1980." The reason for this change is that Zinkl et al. (1980) provides updated information.

Page 49 - In paragraph 1, line 4, delete "(USDA 1968)" reference. This reference is extraneous.

Page 50 - In paragraph 4, line 3, change reference from "Union Carbide 1969" to "Dolinger and Fitch, undated." Dolinger and Fitch (undated) provides updated information.

Page 50 - In paragraph 5, line 5, change reference from "Johansen (1959)" to "Atkins et al. (1981)." Atkins et al. (1981) provides updated information.

Page 53 - In paragraph 2, line 2, change reference from "Zinkl et al. (1979)" to "Zinkl et al. (1980)." Zinkl et al. (1980) provides updated information.

Page 53 - In paragraph 4, delete "by the South Carolina Epidemiologic Studies Center (1979)" and add "(USDA 1981c)" to end of paragraph. These changes correct the citation. The sentence should now read as follows:

In forest openings, actual dermal exposure studies conducted during Maine's spruce budworm spray project showed a total dermal exposure of 10 mg carbaryl for a person (150 pounds) who is 80 percent clothed at the time of application (USDA, 1981c).

Page 53 - In paragraph 6, line 7, change reference from "SCESC 1978" to "SCESC, 1979a." This change corrects the citation.

Page 55 - In second indented paragraph, last line, delete "not." This change corrects the quotation. The sentence should now read as follows:

Alpha-naphthol residues in the residential participants indicated that drift did occur.

Page 55 - In paragraph 1 (first paragraph following indented quotation), last line, change reference from "SCESC 1979" to "SCESC, 1979a, 1979b." This change corrects the citation.

Page 56 - In paragraph 3, first line, change reference from "SCESC 1978, 1979" to "SCESC, 1979a, 1979b." This change corrects the citation.

Page 60 - In paragraph 3, line 17, change reference from "Lijinsky and Taylor 1977" to "Lijinsky and Taylor, 1976." This change corrects the citation.

Page 61 - In paragraph 5, delete sentence 4. The reason for this deletion is that a new label was approved by EPA in April 1985 that expands previous site restrictions.

Page 62 - In paragraph 6, lines 3 and 4, delete "Steelman et al. 1975" reference. The reference is one of several and is extraneous.

Page 64 - In paragraph 3, line 4, delete "Abdalla et al. 1965" reference. The reference is one of several and is extraneous.

Page 64 - In paragraph 3, line 7, change reference from "EPA 1969" to "Meister, 1983." The reason for this change is that Meister (1983) provides updated information.

Page 65 - In paragraph 4, line 3, delete "Pearce 1970" reference. The reference is one of several and is extraneous.

Page 65 - In paragraph 4, line 9, change reference from "Johansen 1959" to "Atkins et al., 1981." The reason for this change is that Atkins et al. (1981) provides updated information.

Page 69 - In paragraph 7, delete first sentence. This statement was inadvertently carried forward from a working draft and is extraneous.

Page 85 - Delete the following references because they are no longer cited in the text:

Abdalla et al., 1965.

Atherton, 1977.

Page 85 - Insert the following references:

Abrahamsen, L. H. and M. Jerkofsky.
1981. Enhancement of varicella-zoster virus replication in cultured human embryonic lung cells treated with pesticide carbaryl. App. Environ. Microbiology 41(3): 652-656.

Brookman, D. H., C. Chopna, D. J. Ecobichon, C. Y. Kana, L. Ritter, and J. Thorsen.
1984. Assessment of the potential of insecticides, emulsifiers, and solvent mixtures to enhance viral infection in cultured mammalian cells. App. Environ. Microbiology 47(1): 80-83.

Page 86 - Delete the following reference because it is no longer cited in the text:

Chemagro, 1968.

Page 86 - Change title of Chevron Chemical Co., 1973, from "Orthene insecticide--environmental impact report" to "The impact of Orthene on the environment."

Page 86 - Change report number of Chevron Chemical Co., 1975, from "75238-13-2/75" to "75238-13-R1 10-75."

Page 87 - Delete the following references because they are no longer cited in the text:

Environmental Protection Agency, 1969.
Environmental Protection Agency, 1975.

Page 87 - Change authors of Doane and Hitchcock, 1964, from "Doane, C. C. and J. W. Hitchcock" to "Doane, C. C. and S. W. Hitchcock."

Page 87 - Insert the following reference:

Elespuru, R., W. Lijinsky, and J. Setlow.
1974. Nitrosocarbaryl as a potent mutagen of environmental significance. Nature 247:386-387.

Page 88 - Delete the following reference because it is no longer cited in the text:

Fishbein, 1978.

Page 89 - Delete the following reference because it is no longer cited in the text:

Innes, et al, 1969.

Page 89 - Change title of Heimpel, 1971, from "Safety of insect pathogens (in man and vertebrates)" to "Safety of insect pathogens for man and vertebrates."

Page 90 - Delete the following references because they are no longer cited in the text:

Johansen, 1959.
Kondakov, 1963.

Page 91 - Delete the following reference because it is no longer cited in the text:

Marston and Voronina, 1976.

Page 91 - Insert the following reference:

Lijinsky W. and C. Winter.
1981. Skin tumors induced by painting nitrosoalkylureas on mouse skin. J. Cancer Res. and Clin. Oncol. 102:13-20.

Page 92 - Delete the following reference because it is no longer cited in the text:

Pearce, 1970.

Page 93 - Change authors of Richmond et al., 1979, from "Richmond, M. L., C. J. Henny, R. L. Floyd, R. W. Marnan. D. M. Finch, and L. R. DeWeese." to "Richmond, M. L., C. J. Henny, R. L. Floyd, R. W. Mannan, D. M. Finch, and L. R. DeWeese."

Page 93 - Insert the following reference:

Schmidt, N.
1983. Effect of the pesticide carbaryl on replication of human and simian varicella viruses. Infection and Immunity 39(3): 1485-1487.

Page 94 - Delete the following references because they are no longer cited in the text:

Staples et al., 1976.
Steelman et al., 1975.

Page 94 - Change date and title of SCESC, 1978, from "1978. Measure of exposure to the carbamate carbaryl: Maine carbaryl study, 1978" to "1979a. Measurement of exposure to the carbamate carbaryl: Maine carbaryl study, 1978."

Page 94 - Change date and title of SCESC, 1979, from "1979. Measure of exposure to the carbamate carbaryl: Maine carbaryl study, 1979" to "1979b. Measurement of exposure to the carbamate carbaryl: Maine carbaryl study, 1979."

Page 95 - Delete the following reference because it is no longer cited in the text:

U.S. Department of Agriculture, 1968.

Page 95 - Change the booklet number of Union Carbide, 1968, from "IOG-0049A" to "ICG-0449A."

Page 96 - Delete the following reference because it is no longer cited in the text:

U.S. Department of Agriculture, 1983.

Page 96 - Change title and report number of Wegner, 1970, from "Bilarcil (R) (Bay 2349) clinical trials 1960-1969 . . . . Rept. 225" to "Bilarcid (R) (Bay 2349) clinical experience 1960-1969. . . . Rept. 2225."

Page 96 - Insert the following reference:

U.S. Department of Agriculture.
1981c. Final programmatic EIS, proposed cooperative 5-year (1981-1985) spruce budworm management program for Maine. Northeastern Area State and Private Forestry, Feb. 12, 1981.

Page 97 - Delete the following references because they are no longer cited in the text:

Wilcox, 1973.
Zinkl et al., 1979.

Page F-38 - In paragraph 2, change last word of line 7 from "of" to "or." The sentence should now read as follows:

Also, applications of 0.7 lb a.i. acephate/acre in New York (LOTEL 1975) resulted in non-detectable levels (less than 0.05 ppm) of acephate in the liver or muscle tissue of rodents trapped in the treated area.

Page F-122 - In Table 7, change the carcinogenic potency for trichlorfon from "0.047" to "0.0047." This change corrects a typographical error.

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Appendix J
Comment Letters and Responses to the Draft Addendum to the Final Environmental Impact Statement as Supplemented — 1985

COMMENTS

All comment letters received during the comment period on the Draft Addendum are reproduced here. Each has been assigned a number (based on the order of receipt) as indicated in Table J-1. Within each comment letter, specific comments requiring a response have been assigned a letter. Responses to the comments begin on page J-112.

Table J-1.--List of comment letters

Commenter	Assigned Number
California Office of Planning and Research	7
California State Clearinghouse	5
Commonwealth of Virginia, Council on the Environment (with enclosure from Virginia Department of Agriculture and Consumer Services)	21
Indiana Department of Natural Resources	22
Maryland State Clearinghouse	4
Mid-Cumberland Council of Governments and Development District	10
Missouri Clearinghouse	11
National Gypsy Moth Management Board	12
North Carolina State Clearinghouse	1
North Carolina State Clearinghouse (with enclosure from North Carolina Department of Agriculture	9
Northwest Coalition for Alternatives to Pesticides and National Coalition Against the Misuse of Pesticides (with enclosures)	16
Northwest Coalition for Alternatives to Pesticides	23
Ohio State Clearinghouse	2
Ohio State Clearinghouse	17
Oregon State Clearinghouse	3
Oregon State Clearinghouse	13
Oregonians for Food and Shelter	15
Pennsylvania Clearinghouse	14
Tennessee State Clearinghouse	6
Tennessee State Clearinghouse	8
Tennessee Valley Authority	18
Union Carbide Agricultural Products Company, Inc.	20
United States Environmental Protection Agency	19

Letter 1

FM206 10/25/85

NORTH CAROLINA STATE CLEARINGHOUSE
DEPARTMENT OF ADMINISTRATION
116 WEST JONES STREET
RALEIGH NORTH CAROLINA 27611

PGS	
CAH	[illegible]
DRT	CHR
LHT	GRM
OKB	RGS
	TGF
	LDW
File:	Date: 11-1-85

ACKNOWLEDGEMENT OF RECEIPT

MAILED TO

U.S. DEPT. OF AGRI., FOREST SERV., APHIS
GARYN E MOOREHEAD
FEDERAL BUILDING, ROOM 663
HYATTSVILLE, MD 20782

FROM

MS. JEANETTE PRIOR
CLEARINGHOUSE STAFF

PROJECT DESCRIPTION

THE BULK OF THIS DRAFT ADDENDUM IS A PLAIN LANGUAGE VERSION OF APPENDIX F. IN ADDITION, TOXICITY DATA AND CANCER RISK CALCULATIONS H HAVE BEEN CLARIFIED TO MAKE THE RISKS MORE UNDERSTANDABLE.

TYPE - DRAFT ADDENDUMTO FINAL EIS-1985

THE N.C. STATE CLEARINGHOUSE HAS RECEIVED THE ABOVE PROJECT FOR INTERGOVERNMENTAL REVIEW. THIS PROJECT HAS BEEN ASSIGNED STATE APPLICATION NUMBER 86E00000285. PLEASE USE THIS NUMBER WITH ALL INQUIRIES OR CORRESPONDENCE WITH THIS OFFICE.

REVIEW OF THIS PROJECT SHOULD BE COMPLETED ON OR BEFORE 11/25/85.

SHOULD YOU HAVE ANY QUESTIONS PLEASE CALL (919) 733-4131.

Letter 2

RCVD. FOSS 11-4-85

STATE CLEARINGHOUSE
State of Ohio - Office of Budget and Management

30 EAST BROAD STREET • 39TH FLOOR • COLUMBUS, OHIO 43266-0411 • (614) 466-0697 / 0698

US DEPT OF AGRICULTURE, APHIS-PPQ
FEDERAL BUILDING, ROOM 663
HYATTSVILLE MD 20782

Attention: GARY E. MOOREHEAD, S. O. PHONE: (301)436-8261

RE: State Clearinghouse Intergovernmental Review-Application Receipt Letter

Project Title: ENVIRONMENTAL IMPACT STATEMENTS
Project Description: GYPSY MOTH SUPPRESSION & ERADICATION PROJECTS, DRAFT ADDENDUM-FINAL EIS AS SUPPLEMENTED-1985, REFER TO OLD SAI NUMBER 35-445-0011

SAI Number: OH851030-E738-36445
Proposed Federal Funding: $00

Dear Applicant:

The State Clearinghouse has received your notification to apply for federal funds. The review process has begun at the State level and will be completed on 85-12-09.

A State Application Identifier (SAI) number has been assigned to your project. Please refer to this number in all future contacts with the State Clearinghouse and the Area Clearinghouse. This number should also appear on line 3a of the Standard Form 424, as a part of your application.

Sincerely,
Linda E. Wise
Project Coordinator

Letter 3

RCVD. FOSS 11-5-85

OREGON PROJECT REVIEW ACKNOWLEDGMENT

State Clearinghouse
Intergovernmental Relations Division
155 Cottage Street N. E.
Salem, Oregon 97310

OR 851025-029-5

Phone (503)378-3732 or Toll Free in Oregon 1-800-422-3600

Applicant: U.S. DEPT. OF AGRICULTURE
Project Title: Gypsy Moth Suppression and Eradication
Date Received: 10/25/85 (Start of 45-day review period)
PNRS #: OR851025-029-5 BE SURE TO PLACE THIS NUMBER ON YOUR APPLICATION BEFORE SUBMITTING TO FEDERAL AGENCY.

Your project notice has been assigned the file title and number that appear above. Please use it in correspondence and, if applicable, enter it in Block 3A on the 424 form for the project. IN ADDITION, YOUR PROJECT NOTICE MUST BE SUBMITTED FOR REVIEW TO YOUR LOCAL CLEARINGHOUSE.

[ ] FEDERAL GRANT [ ] HUD HOUSING [ ] DIRECT FEDERAL DEVELOPMENT
[ ] ENVIRONMENTAL ASSESSMENT [ ] DRAFT EIS [X] FINAL EIS
[ ] STATE PLAN/AMENDMENT

NOTE: Your project was circulated to state agencies checked below.

ECONOMIC DEV. & CONSUMER SVCS
[ ] Agriculture
[X] Soil and Water
[ ] Economic Development
[ ] Fire Marshal
[ ] Housing
[ ] Labor
[ ] Real Estate

EDUCATION
[ ] Education
[ ] Educ. Coord. Comm.
[ ] Higher Education

EXECUTIVE
[ ] Budget

TRANSPORTATION
[ ] Aeronautics
[ ] Director
[ ] Highway Division
[ ] Historic Preservation
[ ] Parks Division
[ ] Public Transit

NATURAL RESOURCES
[ ] Governor's Office
[X] DEQ
[ ] Energy
[X] Fish and Wildlife
[X] Forestry
[ ] Geology
[ ] Lands
[X] LCDC
[X] Water Resources

HUMAN RESOURCES
[ ] Adult & Family Services
[ ] Children's Services
[ ] Community Services
[ ] Corrections
[ ] Employment
[X] Health
[ ] Mental Health
[ ] Senior Services
[ ] Vocational Rehabilitation

MISCELLANEOUS
[ ] Dev. Disabilities Council
[ ] Extension Service
[ ] Other

State Clearinghouse use only:
State Agency Due Date:____________________
Federal Agency:____________________
County:____________________

Letter 4

OCT 31 1985

MARYLAND
DEPARTMENT OF STATE PLANNING
301 W. PRESTON STREET
BALTIMORE, MARYLAND 21201-2365

HARRY HUGHES
GOVERNOR

CONSTANCE LIEDER
SECRETARY

[illegible]. FOSS 11-7-85

October 28, 1985

MEMORANDUM

Director ✓
Programs ✓
Methods
Coordination ✓
Pesticides
R-9 Pest Coord.
Clerical
MFO
SPFO
DFO
File

TO: Addressees

FROM: Guy W. Hager
Director, Maryland State Clearinghouse
for Intergovernmental Assistance

SUBJECT: State Clearinghouse Project Number 84-1-262
DEIS - Gypsy Moth Suppression and Eradication Projects

The enclosed draft addendum to the Final EIS on the previously reviewed reference subject is forwarded for your information and use. If you desire to further comment on the subject, please contact the U.S. Department of Agriculture within three weeks from the date of this memorandum and send an information copy of such response to this State Clearinghouse. If no response is received within this time period, it will be assumed that your agency has no further interest in commenting on the project, and that the requirements of the established procedures have been met.

Thank you for your attention to this matter.

GWH/cw

Enclosure

Addressees:

Wayne Cawley - DOA
Bruce Gilmore - DNR
William Smith - DSP

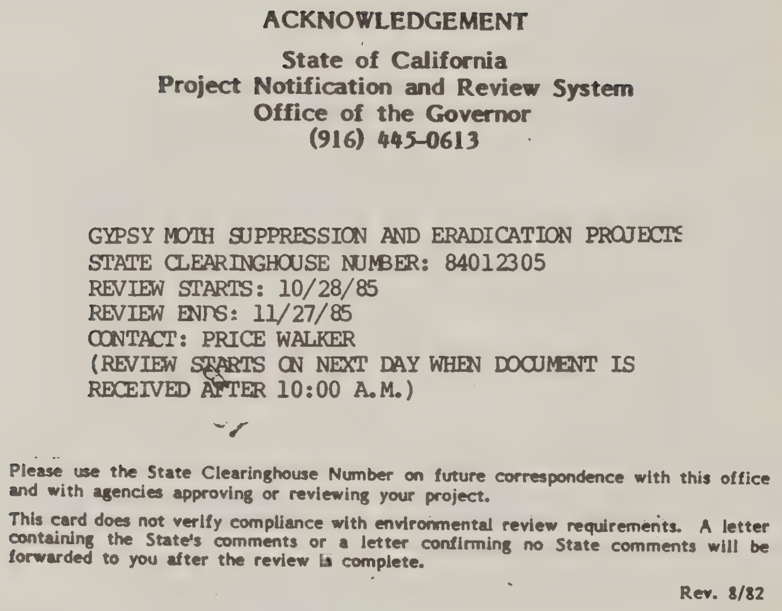
Information Copy:

Thomas N. Schenarts - USDOA

RECEIVED
DIRECTOR, NA-S & PF COOR.
OCT 31 RECD

TELEPHONE: 301-~~[illegible]~~ 225-4490
OFFICE OF STATE CLEARINGHOUSE

Letter 5



ACKNOWLEDGEMENT

State of California
Project Notification and Review System
Office of the Governor
(916) 445-0613

GYPSY MOTH SUPPRESSION AND ERADICATION PROJECTS
STATE CLEARINGHOUSE NUMBER: 84012305
REVIEW STARTS: 10/28/85
REVIEW ENDS: 11/27/85
CONTACT: PRICE WALKER
(REVIEW STARTS ON NEXT DAY WHEN DOCUMENT IS
RECEIVED AFTER 10:00 A.M.)

Please use the State Clearinghouse Number on future correspondence with this office and with agencies approving or reviewing your project.

This card does not verify compliance with environmental review requirements. A letter containing the State's comments or a letter confirming no State comments will be forwarded to you after the review is complete.

Rev. 8/82

Letter 6

CHAIRMAN
JAMES E NAIFEH
VICE CHAIRMAN
KENNETH WESTBROOK

SEC TREASURER
JUDGE ROBERT [illegible] GLASGOW
EXECUTIVE DIRECTOR
ROBERT W BRANDON

POST OFFICE BOX 63
124 WELDON DRIVE
MARTIN TENNESSEE 38237
PHONE 901 587-4213

RCVD. FOSS 11-18-85

November 12, 1985

Mr. Gary E. Moorehead
USDA - APHIS - PPQ
Federal Building, Room 663
Hyattsville, MD. 20782

Dear Mr. Moorehead

Our office is in receipt of the Project Notification from your office concerning Environmental Impact Statement on Gypsy Moth Suppression and Eradication Projects in the United States as Supplemented - 1985. In accordance with OMB Circular A-95 Revised and E.O. 12372, we have reviewed this application and found no conflicts with any planning activities in our area. This is not an approval, but rather a positive clearinghouse review.

This letter should be attached, along with letters from the appropriate State Clearinghouse, to the formal application. If our office, as the Regional Clearinghouse, can be of further assistance, please do not hesitate to contact me.

Sincerely,

Tommy Bradberry

Tommy Bradberry
A-95 Coordinator

TB/bw

SERVING
BENTON-CARROLL-CROCKETT-DYER-GIBSON-HENRY-LAKE-OBION AND WEAKLEY COUNTIES
"PROGRESS THROUGH PLANNING"

Letter 7

Resources Building
1416 Ninth Street
95814
(916) 445-5656
TDD (916) 324-0804

California Conservation Corps
Department of Boating and Waterways
Department of Conservation
Department of Fish and Game
Department of Forestry
Department of Parks and Recreation
Department of Water Resources

GEORGE DEUKMEJIAN
GOVERNOR OF
CALIFORNIA

THE RESOURCES AGENCY OF CALIFORNIA
SACRAMENTO, CALIFORNIA

Air Resources Board
California Coastal Commission
California Tahoe Conservancy
California Waste Management Board
Colorado River Board
Energy Resources Conservation And Development Commission
San Francisco Bay Conservation and Development Commission
State Coastal Conservancy
State Lands Division
State Reclamation Board
State Water Resources Control Board
Regional Water Quality Control Boards

OSS 12-3-85

Mr. Gary Morehead
U.S. Forest Service
Federal Bldg, Room 663
Hyattsville, MD 20782

November 26, 1985

Dear Mr. Morehead:

The State has reviewed the draft addendum to final EIS, Gypsy Moth Suppression and Eradication Projects, submitted through the Office of Planning and Research.

Review of this document was coordinated with the State Water Quality Control Board and the Departments of Conservation, Fish and Game, and Forestry.

None of the above-listed reviewers has provided a comment on this document. Consequently, the State will have no comment to offer at this time.

Thank you for providing an opportunity to review this document.

Sincerely,

Charles K Fellows
for Gordon F. Snow, Ph.D
Assistant Secretary for Resources

cc: Office of Planning and Research
1400 Tenth Street
Sacramento, CA 95814

(SCH 84012305)

Letter 8

RCVD. 12-3-85

TENNESSEE STATE PLANNING OFFICE
1800 JAMES K. POLK STATE OFFICE BUILDING
505 DEADERICK STREET
NASHVILLE, TENNESSEE 37219-5082
(615) 741-1676

LAMAR ALEXANDER
Governor

LEE MUNZ
Executive Director

November 29, 1985

86-0338

Mr. Gary Moorehead
USDA - APHIS - PPQ
Federal Building, Room 663
Hyattsville, MD 20782

SUBJECT: CHTN112985-006 Draft Addendum - Final Environmental Impact Statement as Supplemented - 1985 for Gypsy Moth Suppression & Eradication

Dear Mr. Moorehead:

In accordance with Presidential Executive Orders 12372 and 12416 and with Gubernatorial Executive Order 58, this office serves as the designated State Clearinghouse for federal activities and grants review.

State and local government evaluation of submitted materials has indicated no conflicts with existing or planned activities. Therefore, we are approving this proposal based on the descriptive information made available to us. However, should additional information come to the attention of this office, we may wish to comment further.

This letter should be attached to the application and become a permanent part of the project file. Any involved federal agency should respond in writing to this office if there are problems in complying with this approval. The above State Clearinghouse Identification Number should be placed in the appropriate block on the federal application form.

The appropriate funding agency will now be reviewing our recommendation. If we can be of further assistance, please do not hesitate to contact us.

Sincerely,

Charles W. Brown

Charles W. Brown
Director, State Clearinghouse

CWB:mcp

cc: All Development Districts
All Congressional Districts
U.S. Department of Agriculture

Letter 9

FM208 11/25/85

NORTH CAROLINA STATE CLEARINGHOUSE
DEPARTMENT OF ADMINISTRATION
116 WEST JONES STREET
RALEIGH NORTH CAROLINA 27611

VD. FOSS 12·3·85

INTERGOVERNMENTAL REVIEW COMMENTS

MAILED TO	FROM
U.S. DEPT. OF AGRI., FOREST SERV., APHIS GARYN E MCOREHEAD FEDERAL BUILDING, ROOM 663 HYATTSVILLE, MD 2C782	MRS. CHRYS BAGGETT DIRECTOR N C STATE CLEARINGHOUSE

PROJECT DESCRIPTION

THE BULK OF THIS DRAFT ADDENDUM IS A PLAIN LANGUAGE VERSION OF APPENDIX F. IN ADDITION, TOXICITY DATA AND CANCER RISK CALCULATIONS H HAVE BEEN CLARIFIED TO MAKE THE RISKS MORE UNDERSTANDABLE.

SAI NO 86E00000285 PROGRAM TITLE - DRAFT ADDENDUMTO FINAL EIS-1985

THE ABOVE PROJECT HAS BEEN SUBMITTED TO THE NORTH CAROLINA

INTERGOVERNMENTAL REVIEW PROCESS. AS A RESULT OF THE REVIEW THE FOLLOWING

IS SUBMITTED ( ) NO COMMENTS WERE RECEIVED

(X) COMMENTS ATTACHED

SHOULD YOU HAVE ANY QUESTIONS, PLEASE CALL THIS OFFICE (919) 733-4131.

Letter 9
(continued)

NORTH CAROLINA
DEPARTMENT OF AGRICULTURE

AM G. PARHAM, JR
puty Commissioner

JAMES A. GRAHAM
COMMISSIONER OF AGRICULTURE

RESOURCES, PLANNING, AND DEVELOPMENT
Ray Forrest, Director

November 13, 1985

MEMORANDUM

To: Ms. Chrys Baggett, Director
N.C. Clearinghouse

From: Tom Ellis Tom

Subject: Gypsy Moth, Addendum to the Final Environmental Impact Statement (86-E-0000-0285)

We continue to support this effort and feel that the changes made to the Addendum make this a much more readable document. The supplemental data provided by the Addedum provides a considerable amount of information for use both by program participants and the general public.

TE/jp

attachment

P. O. Box 27647, Raleigh, N. C. 27611 (919) 733-6248

Letter 10

Mid-Cumberland Council of Governments
and Development District

MARSHALL S. STUART, EXECUTIVE DIRECTOR

JOEL PLUMMER, PRESIDENT
WILLIS H. MADDOX, VICE PRESIDENT
ROBERT A. RING, TREASURER

F . FOSS 12-3-85

November 25, 1985

Mr. Gary E. Moorehead
U.S. Department of Agriculture
Federal Building, Room 663
Hyattsville, Maryland 20782

Re: MCCOG/DD #86-129
Draft Addendum to Final EIS on Gypsy Moth Suppression & Eradication in U.S.

Dear Mr. Moorehead:

In accordance with the State and Local Project Review process, and as the Regional Clearinghouse for federal programs, we have reviewed the above referenced project.

Our evaluation reveals no conflict with existing or proposed planning activities. We are notifying you that your proposal is deemed acceptable on the basis of information now available to this office.

We, or other reviewing agencies, may wish to comment further at a later time. This letter should be attached to your application. If we can be of further assistance, please do not hesitate to contact us.

Sincerely,

Maynard Pate

Maynard Pate
Acting Executive Director

MP:pkn

cc: Charles W. Brown, Director
State Clearinghouse
TSPO #86-0338

FIVE HUNDRED AND ONE UNION BUILDING • SUITE L-100 • 501 UNION STREET • NASHVILLE, TENNESSEE 37219 • PHONE (615) 244-1212

Letter 11

RCVD. 11-26-85

John Ashcroft
Governor

John A. Pelzer
Commissioner

State of Missouri
OFFICE OF ADMINISTRATION
Post Office Box 809
Jefferson City
65102

Stan Perovich
Director
Division of General Services

November 22, 1985

Mr. Gary E. Moorehead
Staff Officer
USDA - APHIS - PPQ
Federal Building, Room 663
Hyattsville, MD 20782

Dear Mr. Moorehead:

Subject: 85100036 - Draft Addendum to the Final Environmental Impact Statement on Gypsy Moth Suppression and Eradication Projects in the United States as Supplemented - 1985

The Missouri Federal Assistance Clearinghouse, in cooperation with state and local agencies interested or possibly affected, has completed the review on the above project application.

None of the agencies involved in the review had comments or recommendations to offer at this time. This concludes the Clearinghouse's review.

A copy of this letter is to be attached to the application as evidence of compliance with the State Clearinghouse requirements.

Sincerely,

Lois Pohl

Lois Pohl, Coordinator
Missouri Clearinghouse

LP:cm

Letter 12

NATIONAL GYPSY MOTH MANAGEMENT BOARD

Reply to: North Carolina Department of Agriculture
Plant Industry Division
Plant Protection Section
P.O. Box 27647
Raleigh, NC 27611
(919) 733-6930

OSS 12-9-85

December 3, 1985

Mr. Gary E. Moorehead
USDA-APHIS-PPQ
Federal Building, Room 663
Hyattsville, Maryland 20782

Dear Mr. Moorehead:

RE: Comments on Draft Addendum to the Final Environmental Impact Statement on Gypsy Moth Suppression and Eradication Projects in the United States as Supplemented - 1985

On behalf of the National Gypsy Moth Management Board at the direction of the Executive Committee, I have been instructed to submit a favorable endorsement for the 1985 draft addendum. It is our opinion that readability has been greatly improved and the new addendum should be understandable to most non-technical readers.

It is our hope that this plain language version of the Health Risk Analysis will be submitted to the United States District Court of Oregon and that this version will meet the regulatory requirement for clarity in the court's opinion.

Sincerely,

Howard M. Singletary Jr.

Howard M. Singletary, Jr.
Chairman, National Gypsy Moth
Management Board

HMS/bsw

cc: Executive Committee

Letter 13

OREGON INTERGOVERNMENTAL PROJECT REVIEW

State Clearinghouse
Intergovernmental Relations Division
155 Cottage Street N. E.
Salem, Oregon 97310

[illegible] FOSS 12-9-85

Phone (503)378-3732 or Toll Free in Oregon 1-800-422-3600

CONCLUSIONS

APPLICANT: U.S. DEPT. OF AGRICULTURE

PROJECT TITLE: GYPSY MOTH SUPPRESSION AND ERADICATION

DATE: December 3, 1985

The State of Oregon (and local clearinghouses if listed) has reviewed your project and reached the following conclusions:

☒ No significant conflict with the plans, policies or programs of state or local government have been identified.

☐ Relevant comments of state agencies and/or local governments are attached and should be considered in the final design of your proposal.

☐ Potential conflicts with the plans and programs of state and/or local government:

  ☐ may exist.

  ☐ have been identified and remain unresolved. The final proposal has been reviewed and the final comments and recommendations are attached.

  ☐ have been satisfactorily resolved. No significant issues remain.

A copy of this notification and attachments, if any, must accompany your application to the federal agency.

FEDERAL CATALOG #

NOTICE TO FEDERAL AGENCY

THE FOLLOWING IS THE OFFICIALLY ASSIGNED STATE IDENTIFIER NUMBER:

OR 851025-029-5

IPR #3

Dolores Streeter
Clearinghouse Coordinator

Letter 14

Pennsylvania Intergovermental Council
P. O. BOX 11880 • HARRISBURG, PA. 17108-1880 • (717) 783-3700

RCVD. FOSS 12/10/85

December 5, 1985

Gary E. Moorehead, Staff Officer
ISDA - APHIS - PPQ
Federal Building, Room 663
Hyattsville, Maryland 20782

Dear Mr. Moorehead:

This notice reflects the results of the review conducted through Pennsylvania's Intergovernmental Review Process regarding the Draft Addendum to the Final Environmental Impact Statement on Gypsy Moth Suppression and Eradication Projects in the United States. Copies of these materials were distributed to several of our reviewing agencies; these agencies do not wish to comment.

We appreciate the opportunity to review this document.

Sincerely,

Barbara J. Gontz

Barbara J. Gontz
Project Coordinator
Intergovernmental Review Process

BJG/slk

Strengthening Intergovernmental Relations and Public Decision-making in Pennsylvania

Letter 15

Oregonians for Food and Shelter

1270 Chemeketa Street Northeast
Salem, Oregon 97301
370-8092

“For balance in facts about use of pesticides”

EXECUTIVE COMMITTEE:

CHAIRMAN:
Jerry Bollen
Weyerhauser Company

VICE-CHAIRMAN
Bob Lowry
Willamette Seed & Grain

MEMBERS:
Jim Carnes
International Seed, Inc.

Loyd Smith
Oregon Farm Bureau Federation

Rick Sohn
Lone Rock Timber Company

Jack Brown
Crown Zellerbach

Cathy Baldwin
Willamette Industries

Jim Mischkot
Glass Spray Service

Norm Parker
Western Helicopter

Hobe Jones
Wilbur Ellis

Ken Manning
Oregon Ag. Chem. Assn.

Jim Harris
Oregon Wheat Growers League

Dick Stonex
Longview Fibre Company

December 11, 1985

RCVD. FOSS 12/12/85

Mr. Gary E. Moorehead
Staff Officer
USDA - APHIS - PPQ
Federal Building, Room 663
Hyattsville, Maryland 20782

Dear Mr. Moorehead:

We appreciate the opportunity to offer the following comments on the "Draft Addendum to the Final Environmental Impact Statement as Supplemented — 1985."

Oregonians for Food and Shelter, Inc. (OFS) has identified itself in previous comments to USDA - APHIS on the gypsy moth suppression and eradication projects. Please refer to these previous comments to understand the composition and nature of OFS.

BACKGROUND

OFS has participated in the development of a judicially adequate FEIS for gypsy moth suppression and eradication since 1982. More specifically, OFS intervened in a law suit, on behalf of USDA - APHIS, regarding the propriety of the Final Environmental Impact Statement as Supplemented — 1985 (FEISS).

In that judicial proceeding (OEC, et. al. v. Kunzman, et. al., Civil No. 82-504-RE, hereinafter OEC) OFS argued that the FEISS and its "worst case analysis" were adequate.

The arguments of adequacy put forward by the government and OFS were sustained in all respects save one. The Court found that the worst case analysis in the FEISS (Appendix F) was technically adequate, but that it violated the "plain language" or readability dictates of the Council on Environmental Quality's regulations implementing NEPA (National Environmental Policy Act).

The Court thus invalidated the FEISS for purposes of synthetic chemical insecticide use to suppress or control the gypsy moth.

The draft addendum referenced above is intended to correct this judicially determined deficiency in the FEISS.

Mr. Gary E. Moorehead
December 11, 1985
Page two

COMMENTS

OFS maintained during the OEC proceeding that the FEISS Appendix F was in fact adequate. While Appendix F is technical and detailed, the conclusions reached in the Appendix are explained in plain language in both the summary and main body of the FEISS.

This "tiered" approach to providing adequate information to decision-makers and the public in an FEIS still appears appropriate to us. Nevertheless, the Court held otherwise and the draft addendum has been produced.

We have carefully reviewed the draft addendum (DA) and believe the USDA - APHIS program deserves support for working to produce a "plain language" document.

The DA clearly "demystifies" some of the technical language contained in Appendix F, although we continue to maintain that "demystification" was apparent in other portions of the FEISS.

a The use of graphs and charts in the DA is a welcomed addition. Providing visual reference to readers is a plus. Getting a visual "perception" of the risks at issue will help people understand the problems being discussed. (See, however, figure H-4, which shows a male figure drinking liquid under a spraying operation. This is misleading, if not inane, in terms of reality.)

b However, even though we believe the DA is unnecessary and/or that it is an improvement in the presentation of Appendix F information, one can argue its continuing inadequacy.

Frankly speaking, no one knows if the courts are demanding a true "see Jane run, see Spot run" explanation of information that is by necessity technical in presentation.

The "environmentalists" claim that readability must be universal — this in spite of the fact that most of the few people who zero in on EIS's, the "environmentalists" themselves very often, are highly trained, sophisticated, legally technically competent people.

While we believe the "universal" concept is a Utopian ideal, we also believe that elected and appointed representatives and experts (decision-makers) must be given flexibility in doing the public's work.

Thus, we are placed in a dilemma. We believe the FEISS is adequate in its entirety. We further believe the DA is adequate, if not more than adequate, in addressing the need for a "plain language" Appendix F.

Yet a court, embracing the "universal readability" concept (an impossibility) may disagree.

We provide one simple example.

b

On page H-12, the DA says (first full paragraph on the page):

> "Acephate can cause gene mutations in cells grown in the laboratory. But these effects occur only at dose levels that are so high that they cannot be tolerated by mammals."

A court might ask, "Why not say it this way":

> "Acephate can cause gene mutations in cells grown in the laboratory. However, mutation effects can only be produced in the laboratory because dose levels needed would kill live test animals. Realistically, mutations will not occur."

In other words, why not use "real, short" words for technical terms to lead to "lower" readability levels?

The readability debate is potentially never ending. Yet, substitution of five letter words for ten letter words, inclusion of a glossary of terms, providing document-to-document page references and moving the excellent summary of risk evaluation (p. H-29) to the beginning of the DA may solve many objections.

We truly believe that USDA - APHIS has progressed in production of the DA. We nevertheless fear that courts without scientific expertise may still misread the ability to comprehend the information put forth.

We thus urge USDA - APHIS, in reviewing the DA, to do a worst case readability analysis of its worst case analysis.

Done in this manner, USDA - APHIS may pass judicial scrutiny that, to date, has been careful and cautious, to say the least.

We thank you for your hard work and, again, for this opportunity to comment.

Sincerely,

OREGONIANS FOR FOOD AND SHELTER, INC.

David H. Dietz

David H. Dietz

cc:OFS Confidential List
3PF Board, et. al.

DHD/eac

Letter 16

NCAP
NORTHWEST COALITION for
ALTERNATIVES to PESTICIDES
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RCVD. FOSS 12/12/85

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December 11, 1985

Comments on the Draft Addendum to the Final Environmental Impact Statement on Gypsy Moth Suppression and Eradication Projects in the United States as Supplemented - 1985

The following comments are submitted on behalf of the Northwest Coalition for Alternatives to Pesticides (NCAP) and the National Coalition Against the Misuse of Pesticides (NCAMP).

I. Introduction

a As the title of the addendum (Draft Addendum to the Final Environmental Impact Statement on Gypsy Moth Suppression and Eradication Projects in the United States as Supplemented - 1985) suggests, the public and decisionmakers now have three documents in front of them to wade through as they try to determine the environmental risks posed by federal gypsy moth programs: an EIS, a Supplement (the "worst case analysis" or WCA), and an Addendum (a "readable" version of the WCA).

This trilogy is illegal under the National Environmental Policy Act. The appeals brief submitted on November 26, 1985 to the United States Court of Appeals for the Ninth Circuit (Oregon Environmental Council v. Kunzman) is appended as Attachment 1 because NCAP and NCAMP concur with the appellants that the following are true:

b 1) The entire EIS, not just the WCA, employs difficult, undefined, and convoluted language (and therefore the Addendum merely renders part of the EIS more readable). Brief, pp. 6-12.

c 2) The USDA Forest Service (FS) and Animal and Plant Health Inspection Service (APHIS) did not, as required by NEPA, determine whether they could have done research that would have provided some crucial information for the EIS and WCA and yet would not have been exorbitantly expensive. Brief, pp. 12-18.

d 3) The WCA does not reveal how many people might get cancer from the gypsy moth programs, does not determine the risk associated with multiple applications of spray in a single season, and does not address the cumulative effect of state, private, and federal spraying as required by NEPA. Brief, pp. 18-29.

e 4) The EIS and WCA manipulate and ignore data to conceal health risks. Thus, while appearing to address the issues of carcinogenicity, mutagenicity, teratogenicity, and chronic health effects, the EIS and WCA in fact ignore crucial data (some of which are cited in the EIS and then ignored in the WCA), break rules of logic (including the rules of the risk assessment the WCA has set for itself), science, and integrity in order to arrive at risk estimates that stay somewhere below some threshold of risk for nongenetic effects. As a result, sometimes the EIS can cite a factor of 100x below a No Observable Effect Level (NOEL), sometimes only a factor of 10X below the NOEL, sometimes it switches to a higher NOEL, and sometimes it abandons the NOELs altogether and switches to a Lowest Observable Effect Level (LOAEL). It does whatever is necessary to declare the insecticides safe. For carcinogenicity, the EIS abandons standard risk analysis when necessary to stay somewhere around a "one in a million" cancer risk. Brief, pp. 29-55.

f 5) The EIS claims to consider risks to children and chemically sensitive people and then callously and deceptively applies the same risk factor to children and chemically sensitive people that it had applied to the general population. Brief, pp. 56-58.

g The result of all this is that even if the Addendum were readable, it merely would make an illegal, unscientific, and deceptive document readable. The pronouncement that the insecticides are "safe" may be understandable by the general public, but if the data cited in the EIS and crucial data ignored by the EIS indicate that the insecticides are unsafe, all that has been accomplished is to translate technical deception into plain deception.

In addition, however, the Addendum is flawed in and of itself, as will be noted below.

In the following comments, seven reference terms will be used to refer to four documents or portions of those documents:

Reference term	Document
1. EIS	Final Environmental Impact Statement as Supplmented 1985. USDA Gypsy Moth Suppression and Eradication Projects, all portions except Appendix F.
2. WCA	"Worst case analysis," Appendix F of EIS.

3.	Addendum	*Draft Addendum to the Final Environmental Impact Statement on Gypsy Moth Suppression and Eradication Projects in the United States as Supplemented - 1985.*
4.	H	Appendix H of Addendum.
5.	I	Appendix I of Addendum.
6.	Brief	Brief of Appellants, *Oregon Environmental Council, et al. v. Leonard Kunzman, Director Oregon Department of Agriculture, et al. and John R. Block, Secretary, United States Department of Agriculture, et al.* November 26, 1985. (Attachment 1 of these comments).
7.	O'Brien	Narrative Statement of Mary O'Brien on Behalf of Plaintiffs, *OEC v. Kunzman.* (Attachment 2 of these comments).

II. Comments on Addendum Appendix H: Plain Language Summary of the Health Risk Analysis

OVERVIEW

Ad (Addendum): "All realistic doses to the general public from routine spraying would be at levels deemed safe..." (H-1)

C1 (Comment) 1: There is no safe level for a carcinogen and all four insecticides discussed in the EIS may have carcinogenic effects.

Ad: "The odds of a person getting cancer from routine operations would be no higher than 4 in a million. This is about the same risk as smoking 8 cigarettes in an entire lifetime." (H-2)

C2: No carcinogenic risk analyst would use the term "will be no higher." There are too many uncertainties in the animal data, extrapolation from animal data, and estimation of human exposure to warrant such a statement. Aside from the estimations themselves being low because of underestimations of exposure (Brief, pp. 29-44) and unscientific extrapolation procedures (Brief, pp. 52-55; EIS, Appendix G, Letter 12, pp. 11-12; O'Brien, pp. 14-17), the comparison of compulsory carcinogen exposure (aerial spray programs) to chosen risks (smoking) is unwarranted.

Ad: "It seems extremely unlikely that spraying projects would result in mutations that could be passed to offspring." (H-2)

C3: This is entirely unwarranted in light of evidence the WCA cites: e.g., "...Kiraly et al. (1977) found an increased frequency of chromatid-type aberrations in workers who manufactured trichlorfon....[T]richlorfon is mutagenic in bacteria, yeast and mammalian cells in culture." (WCA, F-14). "[M]utagenicity studies indicate that acephate can induce gene mutations, DNA repair, and sister chromatid exchanges in submammalian or mammalian cells in culture." (WCA, F-12). Despite the WCA contention that this genetic damage would not take place in humans, the H-2 statement is unwarranted.

Ad: "Whenever there is doubt about what might happen when the chemicals are used, this analysis assumes the worst." (H-3)

C4: This is false. For instance, on H-13, the cancer potency of N-nitrosocarbaryl is estimated to be 0.057 for purposes of the analysis of cancer risks (e.g. such as those on H-2). On I-17, however, the Addendum notes that cancer potency based on experiments with female Sprague-Dawley rats would be 7.6. This is 133 times higher than the potency assumed by the WCA.

For dozens of other examples of the falsity of the H-3 statement, see the Brief, pp. 24-59.

Ad: "...[T]here is no evidence that any of the four chemicals have caused cancer in humans at any dose levels." (H-3)

C5: This is intentionally misleading. To my knowledge, no study has ever been undertaken to specifically look for whether cancer has been caused in humans at any dose level by any of the four chemicals. The H-3 statement cannot be made unless another sentence follows or precedes it saying, "There is no evidence that any of the four chemicals have not caused cancer in humans at any dose levels."

HAZARD IDENTIFICATION

Ad: "This hazard analysis always focuses on the lowest NOEL to make sure that risks will not be understated." (H-5)

"Safety factor - Factor applied to lowest NOEL to set safe lifetime dose to humans." (H-7)

C6: This is false. For instance, the lowest NOEL cited for carbaryl on H-11 is 3.125 milligrams per kilogram of body weight per day (mg/kg/day). On page H-12, the Addendum states that a NOEL of 10 mg/kg/day (not 3.125 mg/kg/day) was used to set carbaryl's ADI. Even worse, Table I-1 on I-4 cites a human NOEL of 0.06 mg/kg/day. This is 166 times lower than the 10 mg/kg/day NOEL used to "set a safe lifetime dose to humans." See also Brief, pp. 45-49.

Moreover, whenever estimated doses are above birth defects NOELs, the WCA deceptively and unethically switches to higher NOELs or unidentified LOAELS for purposes of discussing how the four chemicals are "safe." (Brief, pp. 50-52; O'Brien, pp. 9-12).

Ad: "...ADI's are considered to be doses that can be taken safely every day for an entire lifetime. Yet most potential exposures from gypsy moth projects are one-time or short-term exposures. Therefore, comparing the estimated exposures to ADI's and NOEL's might not give a true picture of the risks involved. The error always would be on the side of overstating the risks." (Emphasis added.) (H-8)

C7: This is misleading because the estimate that ADIs are supposedly safe levels for lifetime consumption does not mean they are safe to exceed for short periods of time. They represent a threshold that is supposedly "safe" on any one day and on repeated days. Going over that threshold on any one day might incur adverse health effects.

The statement is also false because it assumes ADIs are accurate. The history of pesticide regulation is replete with estimates of "safe" thresholds being lowered as more is learned. In 1978, a low NOEL for carbaryl was assumed to be 3.125 mg/kg/day based on the beagle birth defects studies. The Addendum cites a 1984 study showing kidney effects in humans at 0.13 mg/kg/day and a NOEL of 0.06 mg/kg/day. The WCA and Addendum references to ADIs are out of date and understate risks.

Ad: "To show how the linear model overstates the effects of low doses, the graph [Figure H-3 on page H-9] includes a curve that is closer to known cancer potencies." (H-9)

C8: The S-curve described as the "More Realistic Potency Slope" in Figure H-3 is a slope that has been found to apply to some, but not all cancer-causing chemicals. Even when an S-curve is known, the place at which it becomes concave is different for different chemicals. There is no basis for generalizing to all chemicals in order to imply that the "linear model overstates the effects of low doses" for the four gypsy moth insecticides. In the absence of information about a particular chemical's carcinogenic behavior, there is no way to know whether a linear slope overestimates the risk at low doses.

Ad: "Because potency slopes and values can be difficult to grasp, this summary also indicates what daily dose of each chemical would result in a 50-percent chance of getting cancer from that chemical. These numbers help compare the potency of the chemicals and are new ways of expressing the data already in Appendix F." (H-9)

C9: The process of telling people how many grams of an insecticide they would have to consume for one out of every two people to contract cancer is outrageous. If "slopes and values" are hard for people to grasp, understandable comparisons among cancer potencies could be made by telling people how many milligrams of an insecticide they would have to consume for one out of every million people to contract cancer as a byproduct of gypsy moth programs.

Telling people how much insecticide they would have to consume for one out of every two people to contract cancer is like telling parents how much asbestos would have to be present in an elementary school for one out of every two children to contract asbestosis. Do you think parents are interested in the amount of asbestos that would kill one out of two children? Do you think the public is interested in what amount of gypsy moth insecticide they have to consume before every other person will get cancer?

Separate your goal of making comparisons of cancer potency understandable from your goal of making carcinogenic pesticides look safe.

Ad: "Cancer is the end result of a multi-step process that starts with mutations (changes) in body cells. Changes in most body cells might lead to cancer. But changes in most cells cannot be inherited by offspring. Cells involved in reproduction--called germ cells--are another matter. Mutations in these cells can be inherited....[I]t may be safe to assume that the risk of heritable mutations would be no greater than the risk of cancer. Cancer and heritable mutations often seem to start in the same way. The main difference is that substances that cause cancer have many more possible targets in the body. While cancer is caused by changes in any cell, heritable mutations are caused only by changes in germ cells. Thus, use of the linear cancer model (which overstates the risk of cancer to estimate the risk of heritable mutations would grossly overestimate such risks." (H-10)

C10: I know of no credible source that quantitatively equates the risk of the multiple steps of cancer with the risk of a mutation occuring in a germ cell. One chemical may easily reach the gonads and be likely to cause heritable mutations but be unlikely to cause cancer. Another chemical may pose a high risk of cancer but be unlikely to cause heritable mutations. Another chemical may cause serious effects via mutations of somatic (non-germ) cells, e.g. in a tissue controlling hormone regulation. A chemical's cancer risk does not place an upper limit on that chemical's mutagenicity risk.

Ad: "All the insecticides under consideration are currently registered by the Environmental Protection Agency for the control of gypsy moth larvae. This means that, in EPA's judgment, available studies indicate that none of these chemicals will cause unreasonable adverse effects in people or the environment when properly used." (H-10)

C11: The following sentence (or a variant thereof) needs to follow the first sentence in the above statement: None of the four insecticides are unconditionally registered by EPA, which means that EPA has either not yet gone through the insecticide's files to determine what additional tests are needed to meet the standards set by Congress in 1972 or, if the files have been examined, that all newly required tests have been submitted to and evaluated by EPA to determine the restrictions that will be put on use of the insecticide when it is fully registered.

Moreover, the second sentence should be followed by the risk-benefit meaning that EPA gives, by law, to the term "unreasonable adverse effects": "The term 'unreasonable adverse effects on the environment' means any unreasonable risk to man or the environment, taking into account the economic, social, and environmental costs and benefits of the use of any pesticide." (Emphasis added.) (Environmental Protection Agency. November 1978. The Federal Insecticide, Fungicide, and Rodenticide Act as Amended).

What this means is that whether cancer or birth defects or kidney damage or any other effect found to be caused by any of these four insecticides is "unreasonable" can only be decided after the effect(s) has been viewed in light of the economic or other benefits that are projected to result to someone from the use of the insecticides.

Since the plain language meaning of "unreasonable adverse effects" is thus not the legal meaning of the term as used by EPA, the plain language requirement of NEPA dictates that the Addendum define the term as a risk-benefit term, not a straight safety term. A readability test would surely indicate that most people reading the sentence on

H-10 would think it meant the EPA thinks these pesticides are safe when properly used. And that isn't what EPA means.

Ad: "The acceptable daily intakes, lowest known no-observed-effect levels, acute lethal doses, and 50-percent cancer probability doses, are compared in Table H-1." (Emphasis added.) (H-10)

C12: This is false. See Comment 6.

Ad: "Based on the effects of large, one-time doses, acephate is considered to be a moderately toxic insecticide. Its main health effect is to reduce the level of cholinesterase, an enzyme found in red blood cells, blood plasma, and elsewhere and that is essential to the functioning of the nervous system."

C13: In a study done for and released by the EPA, large, one-time doses to humans of organophosphate pesticides have now been found to cause what appears to be a permanent intellectual impairment characteristic of individuals with documented physical brain injury. The intellectual impairment may include trouble with information, comprehension, math, vocabulary, spelling, and speech sounds perception. (Eldon Savage, Thomas Keefe, Lawrence Moune, James Lewis, Robert Heaton and Leland Parks. 1982. Chronic Neurological Sequelae of Acute Organophosphate Pesticide Poisonings: An Epidemiologic Study. Final Report. Washington, D.C.: U.S. EPA.)

Moreover, with regard to whether acephate is considered to be moderately toxic, no reference has been made to the fact that acephate is known to convert to methamidophos inside plants, insects, fish, sediment, and birds (Bull, DR. 1979. Fate and efficacy of acephate after application to plants and insects. J. Agric. Food Chem. 27(2):268-272. Geen, GH, MA Sussain, PC Oloffs, and BA McKeown. 1981. Fate and toxicity of acephate (Orthene) added to a coastal B.C. stream. J. Environ. Sci. Health. B16(3)253. Zinkl, JR, RB Roberts, PJ Shea, and J Lasmanis. 1981. Toxicity of acephate and methamidophos to dark-eyed juncos. Arch. Environ. Contam. Toxiol. 10:185-192.)

In rats, methamidophos is 100 times as toxic as acephate (oral LD_{50} acephate, rats: 700 mg/kg; methamidophos 7.5 mg/kg). The latter is, according to EPA standards, an LD_{50} that is indicative of a "highly toxic" insecticide. The EIS, WCA, and Addendum cannot avoid speaking about methamidophos when discussing acephate toxicity.

Ad: "Acephate can cause gene mutations in cells grown in the laboratory. But these effects occur only at dose levels that are so high that they cannot be tolerated by mammals." (H-12)

C14: I know of no credible source that relates the amount of a chemical applied to cells in mutagenicity assays to dose levels in mammals. In mutagenicity assays, the amount of chemical applied to cells may not be related to the amount of the chemical actually taken up by the cells from the culture medium. Therefore, no direct comparison can be made between dose level for cultured cells and the amount administered to whole organisms.

Ad: "The Environmental Protection Agency used a NOEL of 10 milligrams per kilogram per day as the basis for setting carbaryl's ADI." (H-12)

C15: No reason is given for abdicating to EPA on this matter rather than using the lowest NOEL established for humans (0.06 mg/kg/day - Table I-1 at p. I-4). Moreover, the actual test on which the NOEL of 10 mg/kg/day is based is not cited in the EIS, nor is such a test listed in the WCA summary of established NOELs for carbaryl (F-115, Table 7). See Brief, p. 48.

Although the Addendum provides a new, more complete summary of NOELs for carbaryl (Table I-1 at I-4 -I-6), no test with a NOEL of 10 mg/kg/day is cited except a 7-day rat feeding study. Since ADIs are established on the basis of longterm feeding studies (see, for instance, H-7) this would be an inappropriate test on which to determine carbaryl's ADI.

Ad: "...[T]here has been one study to see if actual exposure to carbaryl has led to birth defects in people." (H-12)

C16: If the Addendum is referring to the Halpin study (cited in the EIS, p. 59), the statement is false. The Halpin study compared the birth defects rate in municipalities where carbaryl had been sprayed (regardless of whether an individual had been exposed) to the birth defects rate in municipalities where carbaryl had not been sprayed (regardless of whether an individual had been exposed to carbaryl). In other words, it did not investigate whether "actual exposure" to carbaryl has led to birth defects in people.

Ad: "...[B]ecause of public concern about birth defects, the analyses for this EIS used the lowest known birth defect NOEL. That NOEL is lower than the one used by EPA [and by the EIS to set the ADI] and is 3.125 milligrams per kilogram per day." (H-12.)

C17: Because of lack of public concern about birth defects, the analyses for this EIS does not use the lowest known birth defect NOEL to set the ADI for analysis. See Comment 6 and Brief, pp. 45-49.

When the EIS does mention the birth defects NOEL, it fails to apply it because the estimated worst case doses to the public and small animals approach or exceed it. See Brief, pp. 50-52, and O'Brien, pp. 9-12.

Ad: "It may be that the results of carbaryl studies using dogs should not be applied to humans. Those studies suggest that dogs might be much more sensitive than other mammals to this chemical." (H-13)

C18: What studies suggest that dogs might be much more sensitive than other mammals to this chemical?

According to the EPA, "A major argument that was used to discount the significance of the dog studies was the proposal that there are differences in the metabolism of carbaryl between dog and man. These differences however, have not been demonstrated." (U.S. EPA. 1984. Guidance for the Reregistration of Pesticide Products Containing Carbaryl as the Active Ingredient.)

Ad: "Based on a review of the literature and use of the linear model, the cancer potency of N-nitrosocarbaryl was found to be 0.057." (H-13)

C19: On I-17, the Addendum produces a "new" literature review that includes Lijinsky's studies which indicate a cancer potency as high as 7.6. Moreover, it is stated that the Eisenbrand study "Caused an abnormally high amount of acute toxicity, thereby lowering the cancer potency." Therefore, as pointed out by plaintiffs in the OEC v. Kunzman trial (April 16-18, 1985), the WCA risk estimate is not in fact a worst case analysis risk estimate at all. Instead of rewriting the WCA, the USDA is choosing to have an inaccurate risk estimate of carbaryl cancer risk (from N-nitrosocarbaryl) in the EIS, the WCA, and Appendix H of the Addendum. Numbers that would increase the cancer risk estimate sit unused in Appendix I of the Addendum.

Despite citing a cancer potency 133 times higher than that used in the original WCA, the USDA proposes that "this document [the Addendum] does not...contain significant new information," and therefore does not consider it to be a supplement under NEPA. (Addendum at viii.) What would qualify as significant new information?

Ad: A human would have to ingest 260 grams of carbaryl in order to have a 50% chance of contracting cancer. (H-13)

C20: This is absurd. See Comment 9.

Ad: "Using the linear model, the cancer potency [of 4-chloroaniline, a metabolite of diflubenzuron] was found to be 0.019." (H-14)

C21: The cancer potency of 4-chloroaniline was not determined in the EIS or WCA and no linear model was used. See Brief, pp. 53-54.

Presumably, the Addendum is referring to the discussion of 4-chloroaniline carcinogenicity on I-18-I-21.
The invalidity of that discussion is addressed in Comments 68-70. At any rate, no reference is made to the fact that suddenly material is being utilized from Appendix I rather than the WCA that Appendix H presumably makes readable.

Ad: In order for one out of every two people to contract cancer from exposure to diflubenzuron, "...something like the following would need to occur: Every day of this life, the man would have to eat an entire fish that had been exposed to 5 grams of diflubenzuron." (H-14)

C22: Aren't citizens silly to worry about diflubenzuron? See Comment 9.

Ad: "Trichlorfon is a moderately toxic insectide." (H-14)

C23: This is false. The WCA cites an oral LD$_{50}$ of 144-184 mg/kg/day. (WCA at F-122) According to the EPA, any pesticide with an oral LD$_{50}$ of 50-500 mg/kg/day has a rating of "high" toxicity. In a typical move to downplay the risk that has been documented, Appendix I of the Addendum notes the oral LD$_{50}$ of 144 mg/kg/day (reported by Mobay, the chemical company that markets trichlorfon) but does not mention the fact that this would classify it as a highly toxic insecticide. Instead, it says, "However, based on the most commonly reported LD$_{50}$ values ranging from 400 to 650 mg/kg, trichlorfon can be classified as a moderately toxic insecticide." (I-13).

Ad: "[Trichlorfon's] lowest NOEL is 1.25 milligrams per kilogram per day for reduced levels of cholinesterase in dogs." (H-14)

C24: This is false. Two pages earlier, the Addendum cites a trichlorfon NOEL that is 20% lower: 1.0 mg/kg/day. (H-11).

Ad: "The lowest birth defect NOEL [for trichlorfon] also is from a rat study. This NOEL is 8 milligrams per kilogram per day, which was the highest dose tested." (H-14)

C25: This is false. The Addendum states in Appendix I that the rats were exposed to 8 mg/kg and 80 mg/kg. (I-13)

Ad: A study using hamsters found no birth defects at doses 25 times higher than that [i.e. 8 mg/kg]. (H-14)

C26: What study? The only teratology hamster study cited makes no mention of a 200 mg/kg/dose (i.e. 25x8mga). (I-14)

EXPOSURE ANALYSIS

Ad: "Most of the doses in this assessment are based on actual field studies where carbaryl was used in spraying operations. These studies provide a range of dose levels that actually occurred to both workers and residents during spraying operations that are similar to gypsy moth spraying operations. Because the insecticides are all applied in a similar way and the routes of exposure are expected to be similar for all four chemicals, the carbaryl studies are the best source available for estimating doses in this analysis." (H-18)

C27: The studies referred to used an unpublished, unverified, uncalibrated Union Carbide method of estimating dose of carbaryl by measuring the amount of 1-naphthol in the urine. The Union Carbide method was based on dosing 9 study participants with carbaryl orally and measuring the amount of 1-naphthol for 48 hours. The results indicated that urine collected 6-12 hours after exposure would be the best estimate of exposure, although, according to the authors of the summary of the data, "The small number of participants detracts from the reliability of the findings." (Keil, Julian E., Dr.P.H. [sic] and C. Boyd Loadholt, "Summary of Statistical Analysis of Union Carbide Human Dosing Data." Appendix A in South Carolina Epidemiologic Studies Center. 1978. Measurement of Exposure to the Carbamate Carbaryl. Maine Carbaryl Study, 1978. Draft Report 1 March 1979. [Note: This is the "SCESC 1978" reference in the WCA at F-28]).

Aside from the fact that the "field studies" referred to by the Addendum were unpublished studies using an unpublished, unverified method by Union Carbide of estimating dosage from a gypsy moth spray operation, there are problems with the method itself:

First, the method is based on orally dosing the nine humans and determining the time it takes for 1-naphthol to appear in the urine. Since a major route of exposure for humans and other animals during a gypsy moth spray operation might be dermal (skin) exposure, we have no way of knowing when urine samples should be taken to collect 1-naphthol following dermal exposure. Further, dermal exposure might result in different metabolites from those appearing in the urine with oral ingestion. Therefore, the "field studies'" method of collecting urine for the first 12 hours after a gypsy moth operation may miss collecting the urine at the right time to measure dose or may assay for the wrong metabolite. (Personal communication with Howard Maibach, dermatologist and witness for plaintiffs, OEC v. Kunzman, 1985)

The Union Carbide method apparently lacks essential verification. As stated by Howard Maibach during the OEC v. Kunzman trial, "...[A]ny indirect method such as the naphthol method can only be used reliably when a critical control step...is used; namely, when you take the chemical that you are interested in, namely in this case carbaryl, inject it into the subject or into an appropriate animal as a Rhesus monkey and show that you can account for the injected dose. To the best of my recollection, this had not been done in the 1-naphthol method; therefore, until it is done, it is not possible to know whether those figures are correct.

"...[I]n our particular study,...we used radiolabeled carbaryl. And before we did the skin study, we injected the material into the body and determined the rate for the amount that we could account for, using our radiolabel. Then when we did the skin study, we made the appropriate correction factor. Now if Dr. Wilson [Richard Wilson, defendants' witness who testified that the Union Carbide method is a direct measure of exposure] has done or has available to him the correction factor for the naphthol, then it would be possible to utilize naphthol for a reasonable degree of accuracy. But unless that has become available, that would not be possible." (Emphasis added.) (OEC v. Kunzman Transcript of Proceedings, Volume I, pp. 193-194).

Unless the USDA can adequately answer the charge of Maibach that the Union Carbide is an unverified method and can adequately account for its rejection of Maibach's measurement of 73.9% dermal absorption rate for carbaryl, it fails to insure the professional integrity of its analysis as required by NEPA.

The reason this issue is so important is that nearly all exposure estimates in the WCA are based on the two unpublished field studies using this Union Carbide method.

Ad: "...[A] 10-percent dermal absorption rate is used for all four chemicals even though the estimated absorption rates for each of the chemicals are lower than this." (H-19)

C28: This is false. The only published, direct measurement of skin absorption of carbaryl in human beings found that 73.9% of carbaryl is absorbed through the skin. See Brief, pp. 30-33. The WCA cites the 73.9% absorption rate, but rejects it on the basis that it would contradict results of the two unpublished field studies using the unpublished, unverified Union Carbide method of estimating human exposure to their product, carbaryl. See Comment 27.

Ad: "Gypsy moth spraying is done during the spring--when no fruit and very few vegetables are growing." (H-23)

C29: This is inaccurate. Fruit (strawberries, plums, peaches, apricots, grapes, pears, apples, etc.) is growing in the Willamette Valley of Oregon during May and June when gypsy moth spraying is done. Garlic, onions, leeks, lettuce, peas, beets, asparagus, spinach, cauliflower, cabbage, broccoli, and other green vegetables are growing during that time and have edible portions that would be exposed to the spray. Fruit and vegetables grow year round in California where gypsy moth is found.

Ad: Lifetime dose estimates depend in part on "The amount of chemical a person could be exposed to during each of those days..." (H-25)

C30: The WCA routinely underestimates various parameters of exposure. See Brief, pp. 29-38.

Ad: Lifetime dose estimates depend in part on "The length of time the insecticide remains in meat, on vegetables, or in water (called persistence)..." (H-25)

C31: The WCA routinely underestimates persistence of the insecticides. See Brief, pp. 38-44.

Ad: A person could be exposed 10 times from suppression projects or 6 times from eradication programs in 70 years. (H-25, H-27)

C32: No historical data are cited for this estimate and there is evidence it is an underestimate. See O'Brien, pp. 16-17; Brief pp. 18-29.

Ad: "In humans, this reaction [nitrosation of carbaryl to N-nitrosocarbaryl] could occur only in the stomach, so the only dose that needs to be considered is the dietary dose." (H-27)

C33: Nitrosation of carbaryl can occur in the air before humans breathe or absorb it and, by ignoring this, the EIS, WCA, and Addendum fail to account for all sources of exposure to N-nitrosocarbaryl in a carbaryl spray program. Quoting from a National Research Council paper at the *OEC v. Kunzman* (1985) trial, Dr. Ruth Shearer noted, "'Nitrogen oxides are common environmental pollutants produced by combustion. Four of these compounds have been implicated in forming nitroso compounds; acidic conditions are not required as they are in the stomach. The formation of N-nitroso compounds from nitrogen oxide is usually faster and more extensive than from nucleac [sic] nitric acid.'

"In other words, the reaction is faster and more complete than it would be in the stomach."

"I could find no mention anywhere [in the EIS or WCA] of the risk of formation of nitrosocarbaryl in the air when carbaryl is sprayed in urban areas. All of the discussions concern eating and reaction within the stomach. This would be a minor consideration, in my opinion, with the kind of [gypsy moth] program that we are considering here and the uraban areas frequently involved in gypsy moth spraying." (*OEC v. Kunzman* Transcript of Proceedings, Volume I, p. 73)

Ad: "Carbaryl residues drop to zero in 7 days in meat, in 14 days in vegetables, and in 4 days in water. The analysis used the longest period (14 days) to ensure that the total dose was not understated." (H-27)

C34: This is false. According to data cited in the EIS, carbaryl residues remain longer than 14 days on plants (EIS, p. 49; Brief, p. 42). Other data indicate carbaryl persists 14 *months* in water. (Brief, p. 43)

Ad: "For acephate and trichlorfon, the average lifetime doses were calculated for the two highest combined exposures. The first includes direct exposure to the insecticide during spraying and eating eat [sic] food and drinking water having residues (the direct plus dietary exposure scenario). The second includes an initial direct exposure to insecticide during spraying, as well as additional exposures to residues in food and water (the observer plus dietary exposure scenario)." (H-27)

C35: Lest the reader think, for the first type of exposure, "direct exposure to the insecticide during spraying" means direct exposure to the insecticide spray, it is important to read the WCA at F-30. There the reader learns that estimates for "direct exposure" are based on two types of studies: (a) unpublished studies using the urine analysis method of estimating dosage to residents who lived in a gypsy moth spray area when carbaryl was sprayed, whether they were present, outdoors, indoors, or absent during the spray period and (b) a study that estimated a certain amount of carbaryl exposure to "a bystander"; the EIS then translated that exposure into dose by assuming 10% (not 73.9%) of the carbaryl was absorbed through the skin. The first type of study does not constitute, in the average reader's mind, "direct exposure," and the second does not constitute, for a reader of Maibach's skin absorption study (see Comment 28), an accurate estimate of dosage.

The estimates of exposure to observers were similarly based on urine analyses and 10% absorption of carbaryl. (WCA, F-28)

Ad: "For acephate, the period required for residues to degrade is 20 days in both the realistic and worst case." (H-27)

C36: This is contradicted by the data cited in the WCA. (F-43). The WCA cites some data indicating that 33% to 46% of acephate residues will remain 14 days after application and cites other data that acephate has a half life of 5 to 10 days on plants. A 10-day half-life would mean that residues would degrade to about 5% in 40 days. See Brief, pp. 41-42.

Ad: "The risk of cancer [from diflubenzuron's metabolite, 4-chloroanaline] would come from eating meat or fish, where 4-chloroaniline can be concentrated." (H-28)

C37: This is false. The authors have been told since the public comment period of the Draft EIS as Supplemented that diflubenzuron metabolizes to 4-chloroaniline in soil, water, and (food) plants as well as in animals. Exposure to 4-chloroaniline from these sources must be considered in estimation of the risk of cancer. Brief, pp. 54-55.

Moreover, the WCA assumes diflubenzuron will bioconcentrate with a factor of 1.0. The EPA employs a bioconcentration factor of 50. Brief, p. 55.

Ad: "Dermal doses [for all four insecticides] are multiplied by the dermal absorption rate (10 percent) and then divided by days in a lifetime to get the average lifetime dose from accidents."

C38: The Addendum and WCA illegally apply a 10% rather than 73.9% absorption rate for carbaryl. See Comment 28.

Ad: "Among these 5.4 million people [who could be affected by gypsy moth suppression projects - no estimate is made for those affected by eradication projects] there are individuals or groups (for example, infants) who might be more sensitive than most people to the four insecticides....[T]o be cautious, this group is included in the discussion regarding potential health effects." (H-29)

C39: This group is "included in the discussion" (F-98-F101), but is not accounted for at all. Deliberately and callously, numbers are shifted around so that no allowances are made for chemically sensitive individuals "regarding potential health effects." The same margin of safety that is applied to the general population is applied to chemically sensitive people, but the WCA acts as if it is applying a different standard. See Brief, pp. 56-58.

RISK EVALUATION

Ad: "Based on the yearly number of flights in the past, this suggests that gypsymoth projects could have about 2 aircraft spills every 3 years."

C40: One helicopter crash, mechanical failures, and three helicopter spills occurred during the first two week period of gypsy moth spraying in Lane County, Oregon in 1985.

Ad: "The estimated doses from spraying mostly would be one-time or of short duration. Yet the ADIs are doses that can be safely taken every day for a lifetime. So it would seem reasonable that an estimated dose could exceed the ADI by a small amount without causing harm." (H-30).

C41: What seems "reasonable" to the authors of the Addendum is not necessarily scientifically accurate. It might be "safe" for a person to jump off a one-story roof every day of her life. It would be dangerous for her to jump off a two-story building even one day. See Comment 7.

Ad: "Every dose from routine operations is below the level that could cause birth defects and should be within the margin of safety for the general population in this regard." (H-30)

C42: This is false. If the typical margin of safety of 100x is applied to the carbaryl birth defects NOEL of 3.125 mg/kg/day, every worst case dose and the expected dose of mixers/loaders exceed this margin of safety. Moreover, if the WCA had applied the data regarding mixers/loaders that was present in a study it cites (Maitlen et al. 1982), the worst case dose for mixer/loaders would be above the NOEL for birth defects, even assuming 10%, not 73.9% carbaryl dermal absorption. See Brief, p. 33, including footnote.

Ad: "Estimated doses that are the same as or below the ADI are considered safe and would pose no health risks." (H-30).

C43: This is false. There is no safe dose for a carcinogen or mutagen. All four of these insecticides (at least when metabolized or nitrosated) are considered potential carcinogens and at least two of them are clearly mutagens. In order to approach accuracy, the sentence should read, "Estimated doses that are the same as or below the ADI are predicted to pose no carcinogenic or mutagenic health risks."

Ad: Figure H-7 illustrates a margin of safety with trichlorfon." (H-33)

C44: Would you please make a similar figure using carbaryl and the lowest NOEL of 0.06 mg/kg/day for kidney functioning and the lowest birth defects NOEL?

Ad: "It should be noted that a study of plant and lab workers exposed to acephate found no effects on blood cholinesterase levels." (H-34)

C45: It should also be noted that this was an unpublished study by Chevron Chemical Company, makers of Orthene (acephate).

Ad: "This type of poisoning [severe acute poisoning by an organophosphate] can be treated with atropine sulfate, a common antidote." (H-34)

C46: Antidotes have apparently not been able to prevent the permanent intellectual impairment found to have occurred among people who were poisoned by organophosphates. See Comment 13.

Ad: "The lowest [carbaryl] NOEL is for birth defects in dogs. As discussed in the hazard section, dogs seem uniquely sensitive to carbaryl." (H-34)

C47: This is false. The lowest NOEL cited by the Addendum is for humans and is 52 times lower and is for kidney functioning (I-4, Table I-1). Another NOEL, two times as low, is a systemic toxicity NOEL (I-4, Table I-1). See also Comment 18.

Ad: "[Carbaryl B]irth defect NOEL's from tests using other [non-dog] mammals are much higher, and EPA has concluded that carbaryl will not cause birth defects in humans." (H-34)

C48: There is, to say the least, a great deal of scientific disagreement about "EPA's" statement that carbaryl is not likely to cause birth defects in humans. The EPA's Scientific Advisory Panel met on July 23, 1980 to hear agency representatives give their assessment of the health hazards posed by carbaryl. The Office of Pesticide Programs (OPP) in the EPA had recently decided against initiating a rebuttable presumption against registration (RPAR) of carbaryl, and an agency official had stated that OPP had not found "evidence that carbaryl poses an adverse risk to humans". EPA Office of Research and Development (ORD) teratology researcher Neil Chernoff told the Scientific Advisory Panel that carbaryl is not likely to be a human teratogen.

As reported in the Chemical Regulation Reporter (July 25, 1980, p. 456), "Donna Kuroda, of ORD's Reproductive Effects Assessment Group, told the panel that test evidence showed a teratogenic risk from carbaryl. She said OPP's [Office of Pesticide Program's] evaluation of the teratogenic hazard did not represent the agency's position. Agency staff members told Chemical Regulation Reporter that a working group, including representatives of ORD, OPP, and the agency's offices of Enforcement and General Counsel, voted in late 1978 to recommend that an RPAR for carbaryl be issued. They said Chernoff's assessment of the teratological evidence persuaded OPP that no regulatory action was needed. An ORD staff member told Chemical Regulation Reporter that the Reproductive Effects Assessment Group does not agree that the dog study should be discounted because the dose level produced maternal toxicity. Chernoff ignored evidence that carbaryl in a pregnant animal tends to go to the fetus and remain there longer than in the rest of the body, the staff member added. Panel mamber David Davis said a positive test result in one species should be considered significant. At Davis' suggestion, the panel voted to recommend that carbaryl labeling be changed to include a warning to women of childbearing age. The panel members also suggested that another teratogenic study in dogs be done." (Emphasis added.)

I quote this long excerpt because it is a misrepresentation to say that "EPA has concluded that carbaryl will not cause birth defects in humans." In fact, the EPA Scientific Advisory Panel proposed that carbaryl products bear the warning: "Women of childbearing age should not be involved in the mixing, loading, or application of carbaryl. Exposure to carbaryl during pregnancy should be avoided." (Chemical Regulation Reporter, September 26, 1980).

Ad: "Disregarding tests that used dogs, the lowest [carbaryl] NOEL is for cholinesterase inhibition, and the margin of safety with respect to it is 50. So it is unlikely that the mixer/loaders would have any ill effects." (H-34-H-35)

C49: This is false. The realistic mixer/loader dose approaches the NOEL (with no safety factor) of 0.06 mg/kg/day for kidney functioning and all worst case doses that include a dietary component exceed the NOEL. Compare the 0.06 mg/kg/day NOEL from Table I-1 at I-4 with Table 9 at F-124.

Ad: "[A]ssuming that carbaryl can cause birth defects in humans, the worst case doses might pose some risk of birth defects to sensitive individuals. These doses would be about 1,000 times below the lowest non-dog NOEL's for birth defects." (H-35)

C50: Two deliberately misleading statements are made here: (1) If the worst case dose poses some risk of birth defects to sensitive individuals, it does so equally to women of the general population, because the WCA employs no larger safety factor for chemically sensitive populations than for the general population. See Comment 39. Brief, pp. 56-58. (2) While busily reassuring us that worst case doses to sensitive individuals "would be about 1,000 times below the lowest non-dog NOEL's for birth defects," the Addendum fails to note that worst case doses to the general population are only about 18-27 times below the dog birth defects NOEL, which is the readable interpretation of Table 9, F-124.

Ad: "There is a one in a million chance that...a spill [of insecticide into a creek] would occur." (H-35)

C51: The number of spills expected in gypsy moth programs are underestimated. See Comment 40. In Oregon, the 1983 spruce budworm suppression program in northeastern Oregon experienced a spill that killed all life forms for 22 miles of Willow Creek.

Ad: "...[T]here is a common antidote [to organophosphate poisoning by trichlorfon], so most symptoms could be reversed with prompt medical help." (H-36)

C52: The symptom of permanent intellectual impairment could probably not be reversed. See Comment 13.

Ad: "The numbers in this table [H-9] are weighted lifetime [cancer] risks for all people who are exposed." (H-37)

C53: In the weighted cancer risk, the "worst case" analysis accounts for 0.18% of the risk and the "realistic" exposure accounts for 99.82% of the risk (WCA, F-81). It would be much more informative to prepare a table of "worst case" cancer risk and "realistic" cancer risk just as "worst case" and "realistic" figures for nongenetic effects are prepared (Tables 8-11, WCA F-126).

Ad: On page H-37, N-nitrosocarbaryl's cancer risk in Table H-9 is given based on the WCA potency of 0.057 which the Addendum now rejects as invalid. (I-17). Only in a footnote is mention made of the fact that "If the higher cancer potency discussed on page H-13 were used, all the cancer risks would still be less than 1 in a million." (H-37)

C54: Why is an invalid N-nitrosocarbaryl cancer potency being used for the summary table of carbaryl cancer risk? Moreover, the "higher cancer potency [i.e., 3.6] discussed on page H-13" is merely an average of a variety of cancer potencies that have been found for N-nitrosocarbaryl, the highest of which is 7.6. This latter is the cancer potency that ought to be employed in a proper WCA.

Ad: A discussion of the meaning of cancer risk for carbaryl (from the formation of N-nitrosocarbaryl) in terms of people is given on p. H-38 and H-39.

C55: This entire discussion uses the invalid cancer potency. See Comment 54.

Ad: "The potential cancer risk from diflubenzuron comes from eating meat or fish containing 4-chloroaniline, a breakdown product of diflubenzuron." (H-39)

C56: False. See Comment 37.

Ad: "The cancer risk from exposure to trichlorfon was calculated for members of the general public with the highest exposure. These are persons who receive a direct application and who then eat and drink food and water containing spray residues." (H-39)

C57: This is false. In the WCA, the category "General Public - Direct" does not mean any direct application. See Comment 35.

Ad: "...[C]arbaryl is only weakly mutagenic..." (H-39)

C58: This is an inadequate treatment of carbaryl given that it is the Addendum's total statement regarding carbaryl under the topic "Heritable Mutations." Carbaryl happens to be the only one of the four insecticides that has been shown to cause chromosome damage in whole animals. Moreover, two epidemiological studies dealing with sperm counts of workers formulating carbaryl are regarded by EPA as suggestive evidence that "carbaryl may have the potential to act as a germ cell mutagen." (Chemical Regulation Reporter, December 19, 1980, p.1224.)

III. Comments on Addendum Appendix I: Clarification of Information About the Toxicity of Acephate, Carbaryl, Diflubenzuron, and Trichlorfon

Ad: "Acephate has been shown to be weakly mutagenic in studies with bacteria, fungi, and mammalian cells in culture." (I-2)

C59: This is false. The WCA indicates that acephate was "very positive" in a sister chromatid exchange assay in hamster ovary cells and "positive" with yeast. (WCA, p. F-13)

Ad: "Ingestion [by humans] of 2.8 mg/kg of carbaryl (Sevin formulation) resulted in epigastric pain and sweating." (I-3)

C60: The Addendum cites 3.125 mg/kg/day as the "Lowest NOEL" for carbaryl. (H-11)

Ad: "Subchronic and acute exposure to carbaryl has resulted in decreased levels of cholinesterase and decreased reabsorption by proximal tubules of the kidney in test animals and humans....Both these toxic effects are transitory and have not resulted in permanent physiological damage to the exposed individuals. (Harry, 1977, and Wills et al., 1968)." (I-3)

C61: The Addendum statement is in fact contradicted by the only published reference it cites. The "Harry 1977" citation for the Addendum statement is an unpublished Union Carbide review. The "Wills 1968" reference is a published study that administered 0.06 mg/kg/day and 0.12 mg/kg/day carbaryl to humans. These are very low doses, the higher one 26 times below the "lowest NOEL" cited by the EIS and at or below the estimated worst case dose to the public that includes a dietary component (WCA, F-124, Table 9). Wills et al. make the following comment regarding the 0.12 mg/kg dose: "Because these experiments lasted for only 6 weeks, we cannot say whether the decrease in the ability of the proximal convoluted tubule to reabsorb amino acids might not have become more serious or more persistent with more prolonged exposure to carbaryl. We cannot conclude, therefore, that the higher dose [i.e. 0.12 mg/kg/day] is a safe one." (Emphasis added.)

The Addendum would be more accurate to say that "Very low doses of carbaryl have resulted in decreased reabsorption by proximal tubules of the kidney in an experiment with humans. This toxic effect lasted throughout the six weeks of oral administration of 0.12 mg/kg/day carbaryl, and the reabsorption had not completely returned to pretreatment levels 16 weeks after administration of the carbaryl ceased. The authors note that more prolonged exposure to this low dose might have resulted in more serious or more persistent kidney damage."

That's a different, more accurate rendering of the data than provided by the Addendum.

Ad: On I-7, the Addendum discusses a study (Murray et al., 1979) in which a maternal NOEL for mice less than 1,166 mg/kg/day, a teratogenic NOEL for mice greater than 1,166 mg/kg/day, and fetotoxic effects for mice at 1,166 mg/kg/day were reported.

C62: Was any dietary dose but 1,166 mg/kg/day administered? If not, the conclusions are absurd. If so, why does the Addendum not mention them? The report of the study as it stands is not readable.

Ad: Under "Viral Enhancement," the Addendum notes that a health advisory panel appointed by the Maine Bureau of Forestry to review data on Sevin-4-oil and viral potentiation, concluded that "no uninformed, unconsented human exposure occurs during a forest spray operation." The Addendum cites the Abrahamsen and Jerkofsky (1981) study that found Sevin-4-oil to enhance the replication of varicella-zoster virus (chicken pox) in cell culture. (Not mentioned is Rozee's works cited in Abrahamsen and Jerkovsky that found that a variety of commercial pesticide emulsifiers enhance the sensitivity of cultured cells to infection by certain viruses).

The Addendum then goes on to cite a study that could not replicate Abrahamsen and Jerkofsky's and Rozee's results: "In a recent study by Brookman et al. (1984), various insecticides, solvents, emulsifiers, and mixtures thereof were evaluated to determine whether any were capable of enhancing the sensitivity of cultured mammalian cells to infection with vesicular stomatitis virus. The investigation was replicated in three independent laboratories. None of the compounds, including the insecticide Sevin, significantly enhanced the viral infection." At this point, the Addendum ends its discussion of viral enhancement, making no comment on why the Maine Bureau of Forestry's recommendations will be ignored, but implying it is because the results of Abrahamsen and Jerkofsky and Rozee have been shown to have been somehow nullified. (I-9-I-10)

C63: It is clear that scientific disagreement exists here and the correct course of the USDA should be to consider resolving this important question with research. No estimation of viral enhancement research costs is given in the short list of research costs dictated to the USDA by Dow Chemical and used by USDA to claim that filling data gaps or resolving scientific uncertainty would be "exorbitantly expensive." See EIS, p. 40; Brief, pp. 14-16.

At the very least, the Addendum, EIS, or WCA authors might have considered writing to or speaking with Abrahamsen and Jerkofsky or Rozee to see whether they wished to revise their conclusions in light of the Brookman study. I wrote to Kenneth Rozee asking him about the Brookman study and offer the following excerpts from his response:

"We can demonstrate to anyone that the "Toximal class" of emulsifiers (representing many dozens of popular commercial emulsifers) are virus enhancers with a simple virus plaque assay on treated as compared to untreated cultured cells. Many chemicals are enhancers as pointed out by Dr. Pollack. If you will turn to page 82 of the paper by Brookman et al, you will see Toximul MP8 reported as "toxic" at 100 and 50 ppm in all three laboratories. This contradicts the confidential report by these very laboratories to their employer, the Government of New Brunswick, through their crown corporation 'Forest Products Inc.' which supplied the money for these tests. We obtained these reports (noted in attachments) by a 'circuitous' route from friends in New Brunswick...

"Many other laboratories have repeated our work, Hug; Abrahamson; Pollack and even Thorsen....If you wish, you can talk to any of us at a meeting, or if you are in the Halifax area, please come to our laboratory and we will show you our assays directly." (September 3, 1985 letter from Kenneth Rozee, Head of Microbiology Department, Dalhousie University, Halifax, Nova Scotia).

The USDA has not taken minimal steps to determine whether, in fact, they, like the Maine Bureau of Forestry's health advisory panel, should recommend that "no uninformed, unconsented human exposure occurs during a...[Sevin] spray operation."

Ad: "...[T]he literature shows that N-nitrosocarbaryl only can form under conditions similar to those found in the human stomach--not in the air or on skin surfaces."

C64: What literature? See Comment 33 and then ask Ruth Shearer for the National Research Council paper and any other literature bearing on this issue.

Ad: "A [trichlorfon] teratology study during which pregnant rats were exposed to trichlorfon at the dose level of 8 mg/kg or 80 mg/kg during embryogenesis resulted in malformations and embryotoxicity at the highest dose tested....Based on the absence of terata at 8 mg/kg, the authors suggest that trichlorfon does not pose a danger to humans because the teratogenic dose of 80 mg/kg is 2,500 times the dose likely during a 24-hour exposure period." (I-13)

C65: This is ridiculous reasoning. If, as indicated by the Addendum, only two doses were tested and no birth defects were observed at 8 mg/kg but general edema, hydrocephaly, and meningoencephaly were observed at 80 mg/kg/day, the only conclusion that can be drawn is that birth defects could perhaps also occur anywhere from 9 mg/kg/day to 79 mg/kg/day. Hopefully the experimental expertise of the authors is more professional than their reasoning.

Ad: "Inconsistent results have been reported from various nonmammalial assays examining the mutagenic potential of trichlorfon in bacteria, fungi, plants, and insects." (I-15)

C66: Unless the tests were for precisely the same type of genetic activity, the results should not be characterized as "inconsistent." The reason that a battery of mutagenic assays are administered is because there are so many types of genetic effects and no one "mutagen" is "expected" to cause all types of effects. The plain language interpretation of the Addendum sentence would be that researchers are uncertain as to whether trichlorfon is really a mutagen. This is not the case at all. Trichlorfon has been shown to cause numerous genetic effects in numerous test systems.

Ad: "Therefore, the cancer potency in humans of N-nitrosocarbaryl could range from 0.06 to 7.6 $(mg/kg/day)^{-1}$ depending upon which cancer study was used for the calculation. Using arithmetic average cancer potency values from Lijinsky and Taylor (1976) and Lijinsky and Schmahl (1978), the average cancer potency of N-nitrosocarbaryl would be 3.6 $(mg/kg/day)^{-1}$." (I-17)

C67: The arithmetic averaging of cancer potencies is unsupportable scientifically and the Addendum must not do it. If the cancer potency could range from 0.06 to 7.6 (mg/kg/day)$^{-1}$, then an upper limit of cancer risk should be calculated on the highest of the experimentally determined cancer potencies, and a lower limit on the lowest. By failing to present this range of values for cancer risk, the Addendum fails to present the worst case analysis required by NEPA.

Averaging also unprofessionally denies the uncertainty inherent in cancer risk assessment. By citing an authoritative-looking single number that supposedly denotes "the risk," the authors are less honest than they would be in presenting a range of values within which (or outside of which) the true cancer risk may lie. The absurdity of representing "the" cancer risk by one number is seen by comparing "the" worst case N-nitrosocarbaryl cancer risk from suppression projects in the WCA (i.e. 4 cancers in 100 million at p. F-76) with "the" worst case N-nitrosocarbaryl cancer risk (based on averaging 4 cancer potencies) in the Addendum (i.e. 2 cancers in a million at I-17). "The" worst case cancer risk differs by a factor of 50, depending on which appendix of this total EIS you happen to be reading. If the cancer potencies were not averaged, "the" cancer risk would be a still higher number.

Thirdly, averaging may mask a possible sex difference for N-nitrosocarbaryl-induced cancer. In both Lijinsky studies, the female rats are at higher risk. If in fact human females are also at higher risk, the arithmetic averaging merely underestimates the risk women may face when exposed to a carbaryl gypsy moth program.

Ad: "The cancer potency of 4-chloroaniline therefore could range from 0.0036 to 0.034 (mg/kg/day)$^{-1}$ depending upon which animal study was used to predict cancer in man. The arithmetic average for males of 0.019 (mg/kg/day)$^{-1}$ was used for this analysis." (I-19)

C68: The averaging is unacceptable. See Comment 67.

Ad: "A review of the literature shows that 4-chloroaniline is rarely found in nature. The major metabolites were 4-chlorophenylurea, 2,6-diflurobenzamide, or 2,6-diflurobenzoic acid (see U.S. EPA, 1979, and Nimmo et al., 1984). The principal exceptions were fish and animals, with fish having as high as 60 percent of the total diflubenzuron residue found as 4-chloroaniline (U.S. EPA, 1979)." (I-19)

C69: Apparently this is the Addendum's defense of the WCA failure to consider any human exposure to 4-chloroaniline except meat-eating in its analysis of cancer risk (see WCA, p. F-83; Brief, pp. 54-55; O'Brien, p. 15). The two references cited by the Addendum as the basis for its claim that "4-chloroaniline is rarely found in nature" do not support the claim.

The "U.S. EPA, 1979" reference states, "Table I-5 shows the general metabolic pathway for diflubenzuron in soil, water, plants, and animals. The pathway is common for the production of 4-chloroaniline and 2,6-diflurobenzoic acid in all situations (Opdyck et al., 1976; Phillips-Duphar, December 1, 1975, and December 22, 1975; Booth et al., 1976; Bull and Ivie, 1978; and Nimmo and de Wilde, 1975). The metabolism beyond 4-chloroaniline was reported by Metcalf et al. (1975) in a model ecosystem, to include 4-chloroacetanilide and the 4-chloro-N,N-dimethyl-aniline." (Emphasis added.)

The "Nimmo et al., 1984" reference identifies 4-chlorophenylurea (CPU) and 2.6-diflurobenzoic acid (DFBA) as the chief, first products of hydrolysis in various agricultural soils and hydrosoil. These two products are, according to the authors, "unstable products that are subject to further breakdown and mineralisation." When CPU breaks down, it turns into 4-chloroaniline (as noted in Table I-5 of the U.S. EPA 1979 reference). "The further breakdown of the major soil metabolites 4-chlorophenylurea and 2,6-difluorobenzoic acid will be reported in two subsequent papers in this journal," write the authors in the abstract.

The Addendum claim that 4-chloroaniline is "rarely found in nature" claim must either be backed up with valid data or it should be eliminated. Meanwhile, the EIS continues to fail to disclose important routes of exposure to 4-chloroaniline in its newest cancer risk estimate (I-18-I-20).

Ad: In its calculation of realistic and worst case doses of 4-chloroaniline, the Addendum's Appendix I ("Clarification of Information About the Toxicity of...Diflubenzuron...") continues to apply the WCA bioconcentration factor of 1.0 in fish. (I-20).

C70: This is an underestimation that has been pointed out to the authors before (EIS, Appendix G, page 8 of Letter 12). The EPA 1979 Decision Document on diflubenzuron (the "U.S. EPA 1979" reference in the WCA) assumes a bioconcentration factor of 50 for diflubenzuron in fish.

Ad: The number of annual expected incidences of cancer from gypsy moth programs employing any of the four insecticides is based only on Forest Service records for suppression projects.

C71: This does not account for eradication projects and therefore illegally fails to reveal the total impact of the program proposed in the EIS. Brief, pp. 18-20.

Ad: "In eradication treatments where a second application of a chemical insecticide is applied 7 to 10 days after the first, exposure levels after the second application will exceed doses discussed under the realistic case for threshold effects for all exposure scenarios." (I-23)

C73: What about when three applications of carbaryl are applied as is common according to Appendix E of the EIS? See Brief, pp. 26-27.

Ad: "However, the expected realistic exposure level will be less than 2 times the realistic case dose since some degradation of the first application will have occurred before the second one is applied." (I-23)

C74: This is misleading, given the fact that the half life of diflubenzuron on food plants is one to four or more months, carbaryl persists for months in water, acephate's half life in water may be 50 days, etc. (See Brief, pp. 38-44.)

Ad: "Expected doses [if two applications were made] would equal or slightly exceed the ADI for acephate and trichlorfon when dietary components are included (see tables 8 and 11 of Appendix F)..." (I-23)

C75: This is false. When dietary components are included, expected doses of acephate and trichlorfon would all exceed the ADI. They all equal the ADI for a single application.

Ad: "Expected doses [if two applications were made]...would be lower than the ADI for carbaryl and diflubenzuron (tables 9 and 10 of Appendix F)." (I-23)

C76: The ADI for carbaryl? If carbaryl were applied twice in a program, all expected doses including a dietary component would exceed an ADI properly based 10X below the lowest human NOEL of 0.06 mg/kg/day carbaryl for kidney function. See Comment 6. What about three applications as is common for carbaryl?

Ad: "The expected worst case exposures under the double application eradication approach likely would exceed the ADI only where dietary components are considered for acephate, carbaryl, and diflubenzuron." (I-23)

C77: That's a pretty good guess, given that the expected worst case exposures for acephate, carbaryl, and trichlorfon already exceed the ADI for a single exposure and the worst case exposure for diflubenzuron equals the ADI for a single exposure.

CLARIFICATION OF EXPOSURE INFORMATION

Ad: "Studies of residues of acephate, carbaryl, and trichlorfon on vegetable crops or grass illustrate that initial residues of insecticides range from 1 to 100 ppm depending on the insecticide and type of the vegetation (see, for example, Pieper, 1979; U.S. EPA, 1983; Back, 1961; and Kuhr and Dorough, 1976)." (I-26)

C78: This contradicts data that the EIS itself, NCAP, and plaintiffs in OEC v. Kunzman have cited again and again (see, for example, Brief, pp. 33-34; EIS, p. 49; EIS, Appendix G, p. 5 of Letter 12; O'Brien, pp. 4-5).

Moreover, let's look at the titles of the references the Addendum cites for its "studies of residues of acephate, carbaryl, and trichlorfon on vegetable crops or grass":

The "Pieper, 1979" reference is entitled "Residue analysis of carbaryl on forest foliage and in stream water using HPLC." (Emphasis added). Well, if we're going to cite forest foliage studies, the EIS indicates that initial residues of carbaryl on forest foliage have been found to be as high as 500 ppm 1 day after spraying (EIS, p. 49).

The "Back, 1961" reference is an unpublished Union Carbide Corporation "Memo summarizing surface residues on forest foliage." (Emphasis added.)

The "Kuhr and Dorough, 1976" reference is a book on carbamate insecticides, and the "U.S. EPA, 1983" reference is a residue chemistry chapter in the data evaluation records on trichlorfon.

Taken together, there appears to be no reference on acephate, the first two references are forest foliage references (and if those are applicable to grass and vegetable crops, then the EIS-cited 500 ppm data seem equally applicable), and the last two references may or may not cite data on grass and vegetable crops.

Ad: "These residues [acephate, carbaryl, and trichlorfon] degrade to nondetectable levels wihin 10 to 14 days on vegetation except for grass, which can have detectable residues for up to 28 days (Pieper, 1979)." (I-26)

C79: This claim gets old, too, in light of overwhelming evidence to the contrary (see, for example, Brief, p. 38, 41-43; WCA, p. F-43; EIS, p. 49 and Appendix G, p. 5 of Letter 12; O'Brien, pp. 4-5). The statement is indefensible and should be eliminated.

Ad: "Actual persistence times [of the insecticides in water] depend on many environmental factors, but data from gypsy moth projects indicate that residues do not remain in running water for more than 2 to 6 days (see, for example, LOTEL, 1975, and Pieper, 1979). Persistence can be much longer in stagnant water bodies (Gibbs et al;, 1984), but these are much less likely sources of drinking water." (I-26)

C80: Water has now been divided into "running" and "stagnant." Drinking water from wells, groundwater, and ponds does not exist. The assumption that residues of the insecticides will not be found in drinking or swimming water after 2 to 6 days is false. Carbaryl may persist for months in groundwater (O'Brien, pp. 5-6, Brief, p. 43); acephate may persist for months in water (EIS, p. 43 and Appendix G, p. 5 of Letter 12); diflubenzuron has a half life of two weeks in neutral or mildly acidic water and degrades to 4-chlorophenylurea, the precursor of 4-chloroaniline (U.S. EPA 1979 Decision Document).

IV. Conclusion

h It is clear that many of the above 80 comments cannot be adequately incorporated into the Final Addendum without redoing the EIS with its risk analysis. Incorporating the documented evidence that carbaryl and acephate persist for more than 14 days on food plants, for instance, would require recalculation of the cancer risk posed by these two insecticides and a new discussion of the implications of that cancer risk. Basing the acceptable daily intake of carbaryl on the lowest NOEL for carbaryl (0.06 mg/kg/day) rather than on the unreferenced, high NOEL of 10 mg/kg/day used by the EIS would require a new "Table 9" for carbaryl on "Relationship of expected doses to established exposure thresholds for carbaryl" and a new discussion of the implications of this table in the EIS. The entire EIS should be rewritten in light of these and other comments. The USDA either continues to employ inaccurate data, or it rewrites the EIS. The issue is not academic, because it involves the health of the public and the integrity of gypsy moth decisionmaking.

i The currently separate parts of the larger EIS (i.e., the main body of the EIS, the Appendix F "worst case" analysis, the Appendix H rewrite of Appendix F, the Appendix I presentation of some information from OEC v. Kunzman) are unwieldy, unintegrated, contradictory, and therefore, taken as a whole, unreadable. Crucial data from the EIS, which was originally written without a risk analysis, are not incorporated into the appended "worst case" analysis (Appendix F). Appendix I is tacked on, apparently to acknowledge data presented by plaintiffs in court (OEC v. Kunzman, 1985), but remains unincorporated into the risk analysis of Appendix F, which is, nevertheless, rendered inaccurate by the Appendix I information.

j Many of the 80 comments may be familiar, as they are points that were raised by plaintiffs in the 1983 OEC v. Kunzman trial, in comments on the Draft+Final EIS and the Supplement to the Final EIS, and by plaintiffs in the 1985 OEC v. Kunzman trial. The fact that so many of the valid points raised so long ago remain unaddressed in this newest Addendum must be attributable either to ignorance on the part of the authors as to what has gone before them, or to incredible determination to misrepresent risks posed by the four insecticides. Were the validity of the points raised by NCAP, the OEC v. Kunzman plaintiffs, and others to be allowed to affect the composition of this EIS, WCA, and Addendum, the risk of the insecticides might be considered to be greater than the risk posed by the gypsy moth, at least in some situations. The EIS, WCA, and Addendum, as presently written, however, save decisionmakers from having to make that judgment call, but only at the price of broken logic, ignored data, manipulated numbers, and illegality under NEPA.

These comments are provided on behalf of the Northwest Coalition for Alternatives to Pesticides and the National Coalition Against the Misuse of Pesticides.

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Alternatives to Pesticides

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ATTACHMENT 1

UNITED STATES COURT OF APPEALS

FOR THE NINTH CIRCUIT

Nos. 85-3972, 85-3984

OREGON ENVIRONMENTAL COUNCIL, et al.

Appellants

v.

LEONARD KUNZMAN, Director

Oregon Department of Agriculture, et al.

and

JOHN R. BLOCK, Secretary

United States Department of Agriculture, et al.

Appellees

BRIEF OF APPELLANTS

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IN THE UNITED STATES COURT OF APPEALS

FOR THE NINTH CIRCUIT

OREGON ENVIRONMENTAL COUNCIL; CITIZENS FOR THE SAFE CONTROL OF THE GYPSY MOTH; ELAINE OLSEN AND GLEN OLSEN,

Plaintiffs,

v.

LEONARD KUNZMAN, Director, State of Oregon, Department of Agriculture; STATE OF OREGON, DEPARTMENT OF AGRICULTURE; UNITED STATES DEPARTMENT OF AGRICULTURE; JOHN R. BLOCK, Secretary United States Department of Agriculture; HARRY C. MUSSMAN, Director, APHIS Program, United States Department of Agriculture; R. MAX PETERSON, Chief, United States Department of Agriculture, in their official capacity,

Defendants.

Nos. 85-3972
85-3989

CERTIFICATION REQUIRED BY NINTH CIRCUIT COURT OF APPEALS RULE 13(b)(3)

The undersigned, counsel of record for the Oregon Environmental Council, Citizens for the Safe Control of the Gypsy Moth, Elaine Olsen and Glen Olsen, certifies that there are no known interested parties other than those participating in the case.

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November 26, 1985

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ISSUES PRESENTED FOR REVIEW

1. Is the main text of the EIS written in plain English and can it be understood by the general public?
2. Does defendants' failure to conduct research or properly find the costs of research to be exorbitant violate the National Environmental Policy Act (NEPA)?
3. Does the EIS adequately disclose cumulative impacts as required by NEPA?
4. Do the EIS's misuse and omission of data violate the National Environmental Policy Act?
5. Does the EIS Adequately Disclose Risks to Children and Chemically Sensitive Individuals?

STATEMENT OF THE CASE

On August 30, 1983, this Circuit found an environmental impact statement (EIS) prepared by defendants for their gypsy moth control program in rural areas of the Northeastern United States to be deficient in a number of respects under the National Environmental Policy Act (NEPA). See Oregon Environmental Council v. Kunzman, 714 F.2d 901 (9th Cir. 1983). The case was remanded to the district court for entry of an appropriate order. Clerk's Record (ie, docket sheet) [hereinafter CR] 62. On January 26, 1984, the district court issued a permanent injunction against the aerial application of carbaryl by defendants in populated areas of Oregon until defendants prepared an EIS which complied with NEPA. CR 76. The court retained jurisdiction to review the sufficiency of any new EIS. Id. On January 17, 1984, the federal defendants announced the

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availability of a new draft EIS addressing their national gypsy moth control program. 49 Fed. Reg. 2001.

On March 23, 1984, defendants published in the Federal Register a notice of Final EIS on their gypsy moth program. 49 Fed. Reg. 10963. On May 2, 1984, plaintiffs challenged this 1984 EIS by filing a Motion for Temporary Restraining Order against any federal funding of synthetic pesticides for gypsy moth control under the EIS. CR 95. A hearing was held on May 9, 1984. CR 116. The district court did not act on the Motion for TRO at that time, but instead set a date for trial. CR 116. Trial dates were set and postponed several times during the summer of 1984, while the defendants' spray program went forward. All parties had filed trial briefs on the adequacy of the EIS and on procedural issues by the end of summer, 1984.

On August 23, 1984, defendants published notice of withdrawal of the March 23, 1984, Final EIS, and of their intent to prepare a supplement to the EIS. 49 Fed. Reg. 33472. On December 21, 1984, defendants published a notice of availability of a draft supplement to the withdrawn EIS. 49 Fed. Reg. 49724. A final EIS was issued on March 8, 1985.

A trial took place on April 16-17, 1985, before the honorable James Redden, in the district of Oregon. All parties filed narrative statements and affidavits for their witnesses, supplemented by live testimony, and had an opportunity to cross exam opposing witnesses. The district court ruled on April 26th that the worst case analysis (Appendix F) in the EIS was invalid under NEPA because it was so difficult to read and hypertechnical that it could not be understood by the general public or decisionmakers. ER at 36-39. The court ruled against plaintiffs on the remainder of their NEPA claims. Id. The court enjoined the use of the chemicals discussed in the EIS within the state of Oregon as of April 26, 1985, and enjoined the use of those chemicals on a nationwide basis effective January 1, 1986. Id. On May 21, 1985, the court denied the defendants' request for reconsideration. ER at 44-52.

A. BASIS FOR SUBJECT-MATTER JURISDICTION IN DISTRICT COURT.

Jurisdiction over the instant action was conferred upon the district court by 28 U.S.C. § 1331.

B. BASIS FOR JURISDICTION IN THE COURT OF APPEALS.

Jurisdiction over the instant appeal is conferred upon the Court of Appeals by 28 U.S.C. § 1291. The Court of Appeals has authority, pursuant to 28 U.S.C. § 2106, to affirm, modify, vacate, set aside or reverse the decision of the district court.

C. FINALITY OF ORDER APPEALED.

The court below filed an opinion and order on April 26, 1985, enjoining the defendants from using carbaryl, trichlorfon, acephate, and diflubenzuron as of April 26, 1985 in Oregon, and nationwide as of January 1, 1986. ER at 15, 18. On May 21, 1985, the court denied defendants' Motion for Reconsideration. ER at 44. The court's orders are final, dispose of all claims with respect to all parties, and are properly appealable.

D. TIMELINESS OF APPEAL.

The lower court's order was entered on April 26, 1985 (ER at 15); Plaintiffs filed their notice of appeal on June 25, 1985, within 60 days of the court's original order. Pursuant to 28

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U.S.C. § 2107 and FRAP 4(a)(1), plaintiffs are allowed sixty days for filing their notice of appeal.

E. ATTORNEY FEES.

Plaintiffs seek attorney fees on appeal pursuant to the Equal Access to Justice Act, 28 U.S.C. 2412.

STATEMENT OF FACTS

This case involves the first "worst case analysis" to come before this Court since the seminal decision in Save Our ecoSystems v. Clark, 747 F.2d 1240 (9th Cir. 1984). It presents the question of whether the defendants complied with the National Environmental Policy Act (NEPA) in the EIS they prepared on the impacts of their multi-year program to control the gypsy moth.

The United States Forest Service and Animal Plant Health Inspection Service (APHIS) annually fund numerous state programs involving the aerial application of pesticides, frequently in densely populated areas. See Oregon Environmental Council v. Kunzman, 714 F.2d 901 (9th Cir. 1983). Unrebutted testimony below demonstrated that the entire EIS (Volume II of the Excerpt of Record)(hereinafter EIS) prepared by defendants for this program is so complex and hypertechnical that it cannot be read and understood by decisionmakers or the general public. ER at 86. This Court will have the unenviable task of having to review the document for itself.

Although defendants admit that they are missing significant amounts of information necessary to determine the effects on human health of these pesticides, they refused to conduct any research on those effects, because a Dow Chemical Company official told them research would cost too much. EIS at 40.

Although defendants' EIS purports to address a nationwide, long-term program, the EIS never discusses the potential cumulative, nationwide, long-term impacts on millions of citizens. The EIS deliberately excluded from consideration the cumulative effects of other state and private gypsy moth spray projects. Reporter's Transcript (RT) at 380; ER at 182.

The EIS specifically limited its consideration of health risks to those examined by EPA when it registers pesticides for commercial use, and refused to consider independently any health risks unless they had been considered by EPA. RT at 428.

Although defendants now admit that the chemicals they propose to spray may be carcinogens, mutagens, and may otherwise affect human health, they misuse and selectively ignore data that might reveal the extent of health impacts, establish rules for analysis and then break them, and abandon thresholds of risk when they are unable to manipulate data to make it appear as though those thresholds will not be crossed.

Despite the direct admonitions of this Court in its previous opinion in this case, defendants refused to disclose truthfully the impacts of their pesticides on children and chemically sensitive individuals. ER at 172; RT at 428.

In short, defendants have not truthfully revealed the impacts of the long term, nationwide program for aerial application of pesticides in populated areas that they endorse in the EIS at issue in this case.

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ARGUMENT

I. THE ENTIRE EIS IS NOT WRITTEN IN PLAIN ENGLISH.

Standard of Review: Review in NEPA cases is under 5 U.S.C. § 706(2)(D) (procedures required by law). Lathan v. Brinegar, 506 F.2d 677, 693 (9th Cir. 1974); Northwest Indian Cemetery Protective Ass'n v. Peterson, 764 F.2d 581 (9th Cir. 1985). This standard applies to all issues in this case. This court reviews the district court's conclusion for an "erroneous legal standard" or "clearly erroneous findings of fact." Id.

The duty to write environmental impact statements in plain language is fundamental to NEPA's purposes. The district court erroneously found the main EIS to be comprehensible and did not explain the legal standard it used or the basis for its finding.

A. An EIS That Cannot Be Understood By the Public Is Useless for NEPA Purposes.

The EIS does not comply with the regulations of the Council on Environmental Quality (CEQ), which "Environmental Impact Statements shall be written in plain language" (40 C.F.R. § 1502.8), and "shall be analytic rather than encyclopedic." 40 C.F.R. § 1502.2(a). The EIS also is not "concise, clear, and to the point" as required by 40 C.F.R. §§ 1500.2(b) and 1502.1.[1]

As the court stated in Sierra Club v. Froehlke, 359 F.Supp. 1289 (W.D. Tex. 1973) aff'd in relevant part, reversed on other grounds, Sierra Club v. Callaway, 499 F.2d 982, 994 (5th Cir. 1974) (a pre-CEQ regulation case), under a section of the opinion entitled "Written for the Layman":

> All features of an impact statement must be "written in language that is understandable to non-technical minds and yet contain enough scientific reasoning to alert specialists to particular problems within the field of their expertise." The reason for this standard is the impact statements must assist in rational, thoroughly informed decision making by officials higher up in the agency chain-of-command, including the Congress, the Executive and the general public, some of whom may not possess the technical expertise of those who evaluate the impact and prepare environmental statements. In this regard the present impact statement is deficient.

Id. at 1342-43 (emphasis added)(footnote omitted). See also Silva v. Lynn, 482 F.2d 1282, 1287 n. 6 (1st Cir. 1973) (EIS "incomprehensible" in places); Cf. David v. Heckler, 391 F. Supp. 1033, 1043 (E.D.N.Y. 1984) ("The language used [in medicare notices] is bureaucratic gobbledygook, jargon, doubletalk, a form of officialese, federalese and insurancese, and doublespeak. It does not qualify as English.").

An unreadable EIS is useless for its intended purposes. An EIS must "inform decisionmakers and the public of the reasonable alternatives which would avoid or minimize adverse impacts or enhance the quality of the human environment" (40 C.F.R. § 1502.1), and "to the fullest extent possible: [e]ncourage and facilitate public involvement in decisions which affect the quality of the human environment." 40 C.F.R. § 1500.2(d). Unless an EIS can be understood by decisionmakers and the public, it cannot fulfill these purposes. The CEQ regulations recognize that "[p]ublic scrutiny [is] essential to implementing NEPA." 40 C.F.R. § 1500.1(b). Such scrutiny is impossible if the public cannot read and understand an EIS.

To insure that public scrutiny was meaningful, the CEQ required that EIS's be "written in plain language" (40 C.F.R. § 1502.8), and that EIS's be "concise, clear, and to the point."

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[1] The CEQ regulations are binding on federal agencies and entitled to substantial deference from the courts. Andrus v. Sierra Club, 442 U.S. 347, 358 (1979).

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40 C.F.R. §§ 1500.2(b), 1502.1. Nicholas Yost, who was general counsel to the CEQ when these regulations were drafted and who actually wrote the regulations (ER 190; RT at 349), testified that the regulations were written in response to complaints that EIS's "had become bulky, unreadable, and not really the kind of document that was usable by either of its intended category of readers: the decisionmakers in the government and citizenry representing all sorts of groups." ER 191-195; RT at 350-354. See also 43 Fed. Reg. 55983 (November 29, 1978). Common sense dictates that the public must be able to understand an EIS if it is to participate meaningfully in the NEPA process.

B. The District Court Erred in Ruling That the Main EIS Satisfied Readability Requirements.

The district court correctly ruled that the supposed worst case risk analysis (Appendix F to the EIS) could not be understood by its intended audience and therefore was not legally adequate. See Oregon Environmental Council v. Kunzman, 614 F. Supp. 657, 665 (D. Or. 1985). The court ignored, however, plaintiffs' unrebutted evidence that the main text itself was also not written in plain language and could not be understood by its intended audience. There is no distinction between the readability of the discussion of impacts on human health in the main text and the readability of the discussion of those impacts in Appendix F. See ER at 83; Statement of Shinn at 6. And there is no basis whatsoever in the record for finding such a distinction. Plaintiffs presented unrebutted evidence at trial that the general public cannot read and understand the main text of the EIS (and not just the Appendix). Dr. Mark Shinn, an

educational psychologist and the only expert on readability to testify at trial, performed six different, standard readability tests on the main text as well as Appendix F.[2] ER at 82-83; Statement of Shinn at 5, 6. Dr. Shinn reported: "[r]egardless of the index employed, I found a high degree of consistency in the ratings of difficulty . . . ." Id. (emphasis added). Dr. Shinn concluded that "[t]he FEIS and its appendices . . . would be accurately described as being reading level material suitable for college graduates." Id. (emphasis added). At no time did any witness state that the main text of the EIS could be more easily read or understood than Appendix F. In fact, Dr. Shinn cited two passages from the main text as examples of confusing, technical jargon. ER at 84-85; Statement of Shinn at 7, 8.

Dr. Shinn testified that the average number of years of schooling is 12.3 nationally, but the average reading level for the United States is the sixth grade level. RT at 26. He concluded that he has "no question that the general public would not be able to understand that document as written" (id.), and that the reading levels of even the "best case" samples of writ-

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[2] Dr. Shinn was unable even to determine a reading level under one test, the Spache Readability Index, due to the difficulty of the material. ER at 83; Statement of Shinn at 6. The Flesch Readability Formula "consistently rated these passages at the college graduate level." Id. The Flesch test is well recognized and widely applied; at least 21 states use the Flesch Test to establish minimum readability standards for insurance policies. See, e.g., Hawaii Rev. Stat. § 431.A4-9 (Supp. 1984); Mass. Ann. Laws ch. 175 § 2B (Michie Law. Co-op Supp. 1985); Mont. Code Ann. § 33-15-325 (1983); Nev. Rev. Stat. § 687B.124 (1981); N.J. Stat. Ann. § 17B17-21 (West 1985); N.M. Stat. Ann. § 59A-17 (1983-84); N.Y. Ins. Law § 142(a) (McKinney Supp. 1984-85); N.D. Cert. Code § 26-03.5 (Supp. 1983); ORS 743.350-365 (1983) (Oregon); Tenn. Code Ann. § 56-7-1605 (Supp. 1984).

ing from the EIS "far exceed the estimated reading skills for the U.S. population and the State of Oregon." ER at 84; Statement of Shinn at 7.

Other witnesses, both plaintiffs and defendants, testified to the lack of clarity in the EIS as a whole, even for the sophisticated reader. Plaintiffs specifically asked, in their cross--examination of government witnesses, whether those witnesses would testify under oath that the general public could read and understand the document. Not one would say yes. Defense witness Dr. Wilson, for example (who is the chair of the Department of Physics at Harvard University), stated that the EIS was "not as readable" as he "would like to have seen." ER 189; RT at 223. Defense witness Dr. Calabrese, a professor of toxicology, testified that while the EIS was readable for decisionmakers, "[i]t certainly is not something that I would take to the bathroom with me." ER 161; RT at 261. When asked if the EIS contained a risk analysis for acephate for cancer, he replied: "It probably does. You would have to lead me to it." ER 164; RT at 278. Dr. Brusick, the only defense witness listed as responsible for the readability of the EIS, testified that he believed that a reader with an eighth grade education would require assistance in reading the EIS. ER 159; RT at 325. When questioned at trial about the location of definitions for terms such as "viral potentiation" and "metabolite" in the main body of the EIS, Brusick admitted that no definitions had been included, and remarked, "I'm afraid you could probably mention many terms in here and I wouldn't be able to identify any [definitions]." ER 159, 160; RT at 325, 326.[3] Brusick admitted that in assessing the EIS's readability before it was issued, he consulted no readability experts, employed no readability test, and showed it to no member of the general public. ER 156, 157; RT at 322, 323. Plaintiff witness Dr. O'Brien testified that although she had read the EIS many times, she continued to experience difficulty in locating topics in the EIS because the discussion of health impacts was so scattered and disjointed. ER at 73; RT at 53 (Statement of O'Brien at 18).

The testimony at trial overwhelmingly demonstrated that the entire EIS is overly technical, esoteric and intended to obfuscate rather than explain. Even assuming that readers with -advanced scientific backgrounds may be able to decipher the document after prolonged study, plainly this entire EIS was not written "so that decisionmakers and the public can readily understand [it]." 40 C.F.R. § 1502.8 (emphasis added).[4]

The point is not that an EIS must omit technical discussion or appendices that perhaps require a higher level of sophistication than 50 percent of the U.S. population has (sixth grade reading level). But the EIS itself must be readable by the "general public," Sierra Club v. Froehlke, supra, and facilitate

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[3] In Sierra Club v. Froehlke, supra, the court found an EIS to be inadequate because of (among other understandability problems) it used undefined technical terms. 359 F. Supp. at 1343, n. 215.

[4] The EIS has a striking exception. Its discussion of the bad effects of the gypsy moth itself is almost lyrical, engendering fear and loathing in the reader. EIS at 35. Compare that to the language used to describe possible effects of the pesticides, EIS at 40-41. The latter description is dense, dry, and difficult to read. Defendants know how to use plainlanguage when they want to do so.

"public involvement" and "public scrutiny." 40 C.F.R. §§ 1502.1, 1500.2(d), 1500.1(b). Its intended readership is not just people with a post-graduate or post-high school education, but, as pointed out by Mr. Yost, the "citizenry."

The district court did not explain its reasons for concluding, contrary to all of the evidence at trial, that the main text of the EIS (but not Appendix F) was written in sufficiently plain language to satisfy 40 C.F.R. § 1502.8. OEC v. Kunzman, 614 F. Supp. at 665-65. The court gave no indication of the factual evidence upon which it found the EIS understandable, or the audience and level of sophistication that it believed an EIS must legally reach. The court's conclusion therefore was plainly erroneous and should be reversed. Expert and other testimony from the trial confirm what this Court can see for itself: that the entire EIS's discussion of health risks is confusing, misleading, hyper-technical, and does not provide a clear picture of the actual risks of defendants' program.[5]

II. DEFENDANTS VIOLATED NEPA BY NOT DOING NEEDED RESEARCH.

Defendants have violated their duty to research the health effects of their proposed chemical spraying. An agency is excused from compliance with its duty to do research only if it can demonstrate that the costs are "exorbitant" or the means of acquiring necessary information are beyond the state of the art. Defendants have not made such a showing.

A. NEPA Requires Research Unless Its Costs Are Exorbitant

Section 102(2)(A) of NEPA makes "the completion of an adequate research program a prerequisite to agency action . . . ." Environmental Defense Fund v. Hardin, 325 F.Supp. 1401, 1403 (D.D.C. 1971). This Court has held that NEPA requires defendants to fill information gaps with research whenever missing information is "important," "significant," or "essential" to a reasoned choice among alternatives. Save Our ecoSystems v. Clark, 747 F.2d 1240, 1244 n. 5. In SOS this Court held that one of these same defendants "may be required to do independent research on the health effects" of their proposed chemical spraying. SOS, 747 F.2d at 1248. This Court went on to review the numerous cases finding that NEPA requires agencies to research the impacts of their projects and stated: "The Forest Service does not, and indeed cannot, cite any case which holds that an agency is not obliged to do research to comply with NEPA." Id. at 1249.

Defendants admit that significant data gaps exist. EIS at 39. Rather than seriously considering research to fill those gaps, defendants simply decided to take the easy way out and prepare a worst case analysis.[6] Because researching the

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[5] The Court may wish to compare the EIS with language from a brief filed in this Court, containing the following:

> The duty owing from defendants to plaintiffs in the abstract will vary, under White, relative to the juxtapositions of the real world environmental encasement of the two sides. The concept of causation would seem less plastic.

This Court replied: "Briefs should be written in the English language!" Gottreich v. S.F. Investment Co., 552 F.2d 866, 867 n. 2 (9th Cir. 1977) (exclamation point in original).

[6] The lower court opinion implies that searching "data bases" and having "extensive bibliographies" in an EIS complies with the research duty. OEC v. Kunzman, 614 F.Supp. at 663. This is, however only the beginning of defendants research duty. Such efforts help identify what is known and what is unknown.

environmental impacts of federal projects is so fundamental to NEPA, only if the costs of research are exorbitant or the means of obtaining the information is beyond the state of the art "is the agency freed from compliance and allowed to perform a worst case analysis." SOS, 747 F.2d at 1249 (emphasis added). See 40 C.F.R. § 1502.22(a). Defendants violated this procedure.

B. Defendants Did Not Make a Sufficient Finding of Exorbitance.

Defendants failed to make a reviewable finding in the EIS that the costs of research are exorbitant.[7] Instead, the EIS simply lists the dollar and time estimates for research, and then concludes: "Because of the cost and time involved, a worst case analysis was done ..." EIS at 40. Such a conclusory statement by an agency does not constitute a "reviewable environmental record." Cf. Hanly v. Mitchell, 460 F.2d 640, 647 (2d Cir. 1972) (agency cannot decide against preparing an EIS without making a reviewable environmental record). We cannot know why defendants' concluded the cost and time is excessive, what countervailing factors the agency staff considered, or whether these defendants will ever find research to be a reasonable course of action.

To compound the problem, the listed research "costs" are based solely on an unwritten "personal communication with Jack Warren, Dow Chemical Co." EIS at 40 (emphasis added). Defendants ignored unbiased sources and instead called up the chemical company to ask how difficult it thought health research would be.[8] But defendants could easily have obtained far more credible estimates of the research costs. For example, on page 49 of the EIS defendants cite several residue studies that have already been performed. The actual costs of these studies would be a more credible source for estimating costs than Dow Chemical Company. Testimony at trial established that the costs of already performed research were far below figures such as defendants' unverified, chemical company estimates.[9] For the defendants to base their decision not to research the health effects of toxic chemicals on a phone call from a chemical company is truly shameful.[10]

But the research duty also includes filling the unknown (the data gaps) with new information. SOS, supra.

[7] Of the studies needed to fill the data gaps, defendants claimed at trial that one (but only one) issue of uncertainty (quantification of the risks from heritable human mutations), is beyond the current state of the art.

[8] Estimates of the costs involved were not even mentioned in the draft EIS and draft Supplement. The public had no opportunity to comment on the propriety of relying exclusively on personal communications with industry representatives. Also, the "References Cited" in the back of the EIS do not even mention this supposed "personal communication." This undocumented reference violates 40 C.F.R. § 1502.21, which says that no material may be used and cited "unless it is reasonably available for inspection by potentially interested persons within the time allowed for comment." This is only one of several violations of § 1502.21.

[9] Dr. Shearer testified that she had supervised three research studies involving the mutagenicity and carcinogenicity of a chemical for under $50,000. RT at 106, 107.

[10] Defendants argued post hoc below that one of the "costs" of research would be the damage caused by gypsy moths while such research was underway. But defendants did not seriously consider the use of B.t. to control gypsy moths during the time needed to complete the studies. Defendants argued that "spraying of B.t. (a non-chemical method) alone in heavily infested areas will be inadequate to arrest the spread, much less eradicate the gypsy moth infestation with which Oregon is plagued." CR 181; Defendant's Memo in Support of Summary Judgment at 25. The subsequent success of Oregon's B.t. spraying program in arresting the spread of gypsy moths in a heavily infested area puts to lie such self-serving statements about the lack of alternatives and the cost in terms of time. See Appendix I to this brief.

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Defendants apparently grouped the costs of research for all identified data gaps into one lump sum and then found that total cost to be too great to justify doing any research.[11] But even if the total cost of doing all the research was high, that would not mean that the cost of every individual research project would be exorbitant. To permit defendants to lump all research costs together would allow defendants to avoid doing any research, by simply listing more and more research that could be done. Each gap in needed information has to be evaluated separately.

A sufficient and unbiased estimate of the costs of required research is essential to determining exorbitance. Defendants' estimate of costs is neither.

C. The Lower Court Ignored this Court's Definition of Exorbitance.

This Court clearly stated in SOS that exorbitance is to be measured "in light of the size of the project and/or the possible harm to the environment." SOS, 747 F.2d at 1244, n. 5. Yet defendants asserted below that SOS and the cases cited therein "do not require the agency to compare the total costs of research to the possible harm to the environment." CR 227; Defendant's Reply Brief, at 6. The lower court ignored the direct language of SOS and agreed with defendants. The lower court then mischaracterized plaintiffs' argument in order to reject it. The court said that plaintiffs would require agencies to determine the health costs of illnesses that are unknown, and thus must "have available to it [in advance] all the scientific data that plaintiffs insist it must research." 614 F.Supp. at 664. That is a misstatement of this Circuit's requirement and plaintiffs' position. We already know that the potential harm from these pesticides is that people may develop cancer, birth defects, and other serious diseases. Defendants must evaluate research needs in light of the seriousness of those potential diseases. If defendants believe that a few thousand dollars in research costs are "exorbitant" when compared to the possible prevention of one or more cancers, let them say so. That determination can be then reviewed for arbitrariness. It is not sufficient to say, as the lower court implies, that because cannot calculate the exact likelihood of cancer, it is illogical to compare research costs to potential environmental costs.[12]

As defense witness Dr. Calabrese admitted in his testimony at trial, "If this process [years of aerial spraying] had any rationality to it at all, one would build into the system the capacity to evaluate [the effect on exposed humans] where you have a . . . spray." ER at 162; RT at 274. This Court should break the cycle of aerial chemical spraying in ignorance of the possible severe human health effects that exposure to these

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[11] That defendants should be required to do at least some research in this EIS was admitted by defendants witness Dr. Calabrese, who stated that "a modest small percentage of the total cost [of defendants' proposal] could be a rational way to get some data." ER 162, 163; RT at 274, 275. Dr. Calabrese went on to admit that 5 percent of the cost of defendants' project would not be an exorbitant amount to spend on research. Id.

[12] Defendants also must do research if it is not exorbitant in light of the size of the proposed action. They did not make a finding of exorbitance in the EIS in this respect either, and the lower court did not find that they did. Even if this Court did not require harm to the environment to be considered, comparing research costs to the "size of the project" should not be used to excuse research, but to require it. Pages F-95 and F-96 of the EIS show that over the last 4 years over 13 million people have been exposed to chimical pesticides from defendants' suppression projects alone.

chemicals may cause, and order defendants to do the research NEPA requires.

III. THE EIS DOES NOT ADEQUATELY DISCLOSE CUMULATIVE EFFECTS.

Although defendants propose a nationwide, multi-year program, they fail to analyze the human health effects on a nationwide, multi-year basis. THE EIS claims long-term economic benefits from spraying, but does not disclose the potential long-term health dangers. The EIS also ignores the cumulative health impacts of defendants' own multiple sprayings, and of additional gypsy moth spray projects by states and private individuals. The EIS thus hides the true cumulative impacts on human health.

A. The EIS Does Not Reveal How Many People May Get Cancer from Defendants' Overall Program

Defendants propose to fragment their consideration of human health risks and thereby avoid disclosing the overall number of cancers that may result from their multi-year program. This is directly contrary to 40 C.F.R. §§ 1502.22(b), 1508.7, 1508.25, 1508.27(b)(6) and (7), and this Court's opinion in Thomas v. Peterson, 753 F.2d 754 (9th Cir. 1985).

1. The EIS's formulas and averages do not reveal the total impact of the program.

The chemicals defendants propose to spray over populated areas of the nation are known or potential carcinogens as defendants now admit. EIS at v. The EIS acknowledges that the gypsy moth problem will be around for the foreseeable future, and therefore that government control programs using chemical sprays will span many years.[13] But defendants never disclose how many cancers could occur over the life of the entire proposed program.

Instead, the EIS merely contains mathematical formulas which defendants claim allow readers to calculate the overall number of cancers from spraying. The disclosure problem is that defendants did not bother to apply their formulas to their multi-year program.

First, the defendants provide a formula (on page F-82 of the EIS) for calculating the lifetime risk of cancer per acre sprayed,[14] but do not apply this formula at all. Second, defendants provide a "formula" (on pages F-95 and F-96 of the EIS) for "calculating incidences of cancer per project."[15] But defendants only apply that formula to a single "average" year (based on the number of acres sprayed over the last four years), not to the

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[13] Defendants have been attempting to eradicate the gypsy moth with pesticides in the United States for 35 years (EIS at 6), only to see it expand its territory. EIS at 3. Defendants' intention to suppress the gypsy moth has not changed. EIS at 12, 13. Defendants admit that chemical pesticides remain as one of the preferred methods for controlling gypsy moths. EIS at 11, 1. Thus defendants' chemical spray program will last until the gypsy moth is eradicated, or new suppression technology is developed. EIS at 1.

[14] "For suppression projects, incidents of cancer are calculated as follows (example for carbaryl: 4.84 x 10^{-8} /10 applications x no. of acres treated)." EIS at F-82.

[15] Pages F-95 and F-96 do not contain actual estimates of incidents of cancer, but only examples of how to apply the formulas. Even the examples are, at best, dense. The court below said these pages are "hypertechnical, complex and replete with lengthy equational calculations." OEC v. Kunzman, 614 F. Supp. at 665. "Dr. Richard Wilson, Chairman of the Department of Physics at Harvard University, stated that he was unable to decipher the precise meaning of the passage, but given 15 minutes of study he could probably untangle the message." Id.

full duration of the actual proposal.[16]

By authorizing years of future spraying without revealing the cumulative effects during the reasonably foreseeable life of the program, the EIS hides the bottom line from decisionmakers and the public. As we shall show, this violates NEPA.

2. The district court erred by allowing defendants to fragment their analysis of health risks.

Defendants propose to fragment their analysis by delaying any projection of potential incidents of cancer until site-specific EA's are prepared for individual projects. The court below endorsed defendants' decision not to disclose nationwide impacts for the life of the program, and held that the EIS was intended merely "to outline the risks" associated with particular chemicals.[17] Oregon Environmental Council v. Kunzman, 614 F. Supp. 657, 663, 664 (D.Or. 1985).

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[16] Even the "average year" estimates on pages F-95 and F-96 of the EIS do not provide a basis for estimating the total cancers that may result from defendants' proposed spraying, because the "average year" figures do not include the full extent of defendants' spray program. Page F-95 states that the average numbers for acres and people exposed are based upon only "Forest Service records" of spraying. Thus, these averages do not include the other federal spraying included in defendants' proposal. The Forest Service is primarily concerned with so-called "suppression" spraying. EIS at 12, 13. But this EIS also proposes spraying for "eradication," by defendant Animal and Plant Health Inspection Service (APHIS). EIS at 1, 12, 13. Their spraying is not included in the "average" year.

Also, defendants' "average" numbers of acres is simply taken from an average of the last four years in only fourteen states with no projection of the worst case levels that could be expected in future years. Yet the EIS portrays the gypsy moth as a rapidly spreading problem. The EIS summary lists 27 states that are presently infested with gypsy moths. EIS at iii. The EIS also predicts that the "[n]atural spread of the insect will likely continue . . . to adjacent states," and that the artificial movement of the gypsy moth will continue to spread to "other states where suitable host material exists." EIS at iii, iv. If, as defendants warn, the gypsy moth is on the increase, spraying for suppression and eradication would also be expected to increase. The use of an "average" number of acres for past years is not a "worst case" estimate of the future.

The lower court's ruling is contrary to case precedent and CEQ regulations. These same defendants were told earlier this year that segmenting the discussion of future environmental impacts into small environmental assessments, covering only a portion of their overall future program, is illegal. In Thomas v. Peterson, 753 F.2d 754 (9th Cir. 1985), this Court held that future site-specific EA's would be inadequate for considering the cumulative effects of a Forest Service plan to develop a roadless area over a 20-year period. This Court noted that "consideration of environmental impacts in the decisionmaking process . . . requires that the NEPA process be integrated with agency planning 'at the earliest possible time,' 40 C.F.R. § 1501.2 . . . ." 753 F.2d at 758 (emphasis added). Several other specific regulations also apply.

First, 40 C.F.R. § 1508.7 defines cumulative impacts as:

> the impact on the environment which results from the incremental impact of the action when added to other past, present, and reasonably foreseeable future actions . . . Cumulative impacts can result from individually minor but collectively significant actions taking place over a period of time. (emphasis added).

The EIS in this case was prepared because it is "reasonably

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[17] Defendants' EIS clearly shows that their proposed program is nationwide in scope. For example, defendants state in their EIS, "APHIS eradication projects will be conducted in areas where the insect has been introduced artificially. The artificial introductions may occur in areas throughout the continental United States and Hawaii. EIS at 32. (See also footnote 14, supra.) Plaintiffs simply asked that the number of human cancers be reported for the additional states where spraying is reasonably foreseeable under the life of this EIS.

foreseeable" that spraying will continue for some time to come.

Second, 40 C.F.R. § 1508.27(b)(6), in defining "significance" for purposes of EIS's, provides that an agency must consider:

> The degree to which the action may establish a precedent for future actions with significant effects or represents a decision in principle about a future consideration.

The very purpose of defendants' EIS is to set a precedent for future actions to control the gypsy moth. EIS at 1, 12. And by concluding that the chemicals it purports to examine are acceptable means of achieving its goals, the EIS represents at least a decision in principle about future actions. See EIS at 1 ("The alternative implemented will guide USDA participation in gypsy moth suppression and eradication projects.").

Third, 40 C.F.R § 1508.27(b)(7) states that an agency must consider:

> (7) Whether the action is related to other actions with individually insignificant but cumulatively significant impacts. Significance exists if it is reasonable to anticipate a cumulatively significant impact on the environment. Significance cannot be avoided by terming an action temporary or by breaking it down into small component parts. (Emphasis added.)

In the instant case, defendants plan to spray pesticides over a period of many years. By saying that one will have to look at later site-specific EA's to find the fragments of any analysis, defendants avoid disclosing overall cancer risks and break the impacts down "into small component parts."

At no point will the national decisionmakers on defendants' national, multi-year program be made aware of the cumulative

health effects of defendants' long range proposal to use chemical pesticides. As in Thomas, "Not to require [early estimates of total effects] would permit dividing a project into multiple 'actions,' each of which individually has an insignificant environmental impact but which collectively have a substantial impact." 753 F.2d at 758. Although the CEQ regulations and Thomas were brought to the lower court's attention, the court did not apply them to the facts of this case. The district court's ruling in this case would allow defendants to do just what was ruled illegal in Thomas.

3. The lower court ignored NEPA worst case analysis law.

The lower court held that it would be "unreasonable" for the defendants to estimate the overall number of cancers for the projected life of the multi-year program covered by the EIS because to do so "would be conjectural." OEC v. Kunzman, 614 F. Supp at 664. But EIS's must contain reasonable conjecture. This is especially so in a worst case analysis, which by definition is to discuss risks when there is a high level of uncertainty. As this court stated in Save Our ecoSystems v, Clark, 747 F.2d at 1244, n.9:

> The basic thrust of . . . NEPA is to predict the estimated effects of proposed action before the action is taken and those effects fully known. Reasonable forecasting and speculation is thus implicit in NEPA, and we must reject any attempt by agencies to shirk their responsibilities under NEPA by labelling any and all discussion of future environmental effects as 'crystal ball inquiry.'

The cumulative incidents of cancer must be "formulated on the basis of available information, using reasonable

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projections of the worst possible consequences . . . ." Id. at 1244 (emphasis added). Defendants must at a minimum estimate the amount of time their program will be in effect and the area and population that will be sprayed during that time. This will produce crucial information to national decisionmakers about the cumulative impacts of the gypsy moth control program: How many cancers may be caused by this program over the years? It is no excuse that some degree of prediction and projection may be required to supply this critical information.

4. Defendants are capable of estimating the total incidents of cancer.

The lower court stated: "The EIS cannot estimate the total number of human exposures without the EA's . . ." 614 F. Supp. at 664 (emphasis added). But perfect knowledge of future projects is not a prerequisite to considering their potential cumulative effects. Furthermore, the court simply ignored information already supplied in the EIS, and testimony by defendants' witnesses, upon which the EIS could have projected the "reasonably foreseeable" scope of their proposal and its impacts.

A great deal of information exists from past spraying. First, Appendix E reveals the number of acres sprayed in eradication projects since 1967. Second, the EIS reveals that defendants have records of their use of chemical pesticides going back to 1957.[18] Third, the EIS states that records exist for one of the particular chemicals at issue, carbaryl, since 1962.[19] Defendants' witness Schneeberger admitted at trial that records exist for "suppression" as well as for "eradication" spray projects. When asked: "Do you have any factual historical data showing you what the historical trend has been in suppression projects?" Schneeberger replied,

> Well, we have historical records of, as I described earlier in past environmental impact statements, EA's reports, yearly reports, both our own where we describe the areas you know, the pesticides used and acreage reports. We have the individual states' reports that come out yearly from cooperations participating in the program that identify the areas treated, the acreage and locations. ER 180-181; RT at 378-79 (emphasis added).

Defendants also are able to estimate the future growth and spread of the gypsy moth itself. The EIS shows "the total cumulative defoliation recorded since 1924, particularly the recent rapid increases." EIS at 3. The table on page 2 shows the total acres of defoliation caused by gypsy moths and the EIS on page 3 contains data that show the cyclical nature of the moth's activities, including number of acres defoliated during a cycle. This information, along with information on spraying history, provides a data base from which to estimate the number of acres that may be sprayed in future years. Defendants' EIS proves that they are capable of estimating some kinds of impacts, at least when it is in their interest to do so:

> Significant economic impacts are predicted outside the generally infested area if

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[18] "At its peak use in 1957, more than 2 million acres of forest and forested communities were treated with DDT. During the period of its use, DDT was applied to more than 12 million acres of forest in 9 Northeastern states and Michigan for gypsy moth control." EIS at 6.

[19] "From 1962 to 1977, almost 2 million pounds of this material [carbaryl] were used by Federal and State agencies against gypsy moth . . . ." EIS at 6-7.

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> isolated infestations become permanently established. For example, potential losses in California ranging between $446 million and $457 million for the period 1982 to 1999 have been predicted . . . . EIS at ii (emphasis added).

For economic effects of the moth, defendants' EIS thus "predicts" impacts out 15 years into the future, through the year 1999.

NEPA requires "reasonable forecasting and speculation" for health effects, not just for worst case predictions of economic losses. Defendants possess ample data from which to do such reasonable forecasting. Impacts on human health are at least as deserving of defendants' predictive efforts as dollars. See Thomas v. Peterson, 753 F.2d 754, 760 (9th Cir. 1985).

B. Defendants Did Not Adequately Discuss Other Cumulative Effects of Multiple Exposures.

The EIS also fails to assess the cumulative impacts of the multiple sprayings required in their own eradication projects, and additional spraying in other state and private projects. NEPA requires analysis of total effects in both instances.

1. Defendants' multiple sprayings.

The lower court found that defendants adequately considered the effects of multiple sprayings, based on Dr. Schneeberger's testimony that the health effects of "multiple direct exposures" were taken into consideration. OEC v. Kunzman, 614 F. Supp. at 662. This is incorrect. Dr. Schneeberger in fact admitted that tables 8 to 11, on pages F-123 - F-126 of the EIS, which are used to analyze the health risk to humans, take into consideration only the health effects of a single spraying. ER 179; RT at 370. Yet defendants admit that in their eradication projects, "Up to 3 applications of chemical insecticides may be applied

over a 6-week period . . ." EIS at F-63. The EIS thus admits multiple sprayings will occur during projects, but calculates and displays risks only from a single spraying.

Dr. Schneeberger admitted under examination that the EIS only "provides the mechanism for evaluating any number of applications that you would like." ER 179; RT at 370. In other words, the only way for decisionmakers to inform themselves of the true health risks of defendants' spray programs is to get out a calculator. The main body of the EIS never alerts decisionmakers or the public to this drawback of its tables. On their face, the calculated doses in the tables purport to summarize the actual risks. In fact, the EIS earlier presents a summary table asserting that all "realistic" doses are equal to or below the ADI's. EIS at 17. This graphically pleasing table, with the word "Yes" reassuring the reader that "realistic" doses will not exceed safety levels, is based, however, on a single-application. Tables 8-11 in Appendix F. To understand the importance of this deception, one need only realize that realistic dietary and other doses of the pesticide trichlorfon for the general public on Table 11 (page F-126) are already equal to the ADI. If the EIS on Table 11 had reported the doses from multiple sprayings, they would therefore be substantially above the ADI, because the residues from the first spraying would still be on food plants when second and third sprayings occurred.[20]

The summary of the EIS, a critical section for

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[20] See the discussion of persistence in part IV.A.3. of this brief, infra. Dr. Schneeberger's admissions at trial, quoted above, are to this affect.

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decisionmakers, likewise affirmatively misrepresents risks when it says (based on tables 8-11) that "all realistic doses and many worst case doses for suppression and eradication projects are below [ADI's] and therefore within acceptable margins of safety." EIS at v. This is simply false. To assess risks, decisionmakers and the public would have to ignore this Summary and meticulously recalculate dosages, and their relationships to ADI's and NOEL's, in tables 8-11. Having done these calculations, the reader would still have to guess at the significance of those new numbers for health risks.

2. State and private spraying.

Defendants also refused to consider the impact of their spraying in conjunction with sprayings by state, city and private individuals. Dr. Schneeberger admitted under examination that sprayings by a state, city, or private person were "outside the scope of the EIS." RT at 380-382. Yet defendants freely admit that state and private agency projects may occur in the same area as federal projects, both simultaneously with such projects and in preceding and succeeding years. RT at 385-88 (testimony of Dr. Schneeberger); EIS at 4.

This directly violates 40 C.F.R. 1508.25(2), which requires agencies to consider:

> the impact on the environment which results from the incremental impact of the action when added to other past, present and reasonably foreseeable future actions regardless of what agency (federal or non-federal) or person undertakes such other actions. (Emphasis added).

In summary, defendants failed to discuss the cumulative effect of their own multiple, long-term, nationwide sprayings

in this EIS, and of state and private spray projects. This violates NEPA, regulations of the CEQ, and this Court's opinion in Thomas v. Peterson, supra.

IV. THE EIS MANIPULATES AND IGNORES DATA TO CONCEAL HEALTH RISKS.

The EIS consistently misapplies the data it cites in order to avoid disclosing the risk implied by those data. The EIS presents the very opposite of a worst case analysis: it gives decisionmakers and the general public an optimistic "best case" analysis by glossing over important evidence of likely adverse effects. A detailed analysis of the EIS is necessary to understand how defendants' misuse of data occurs. If criticism of such fundamental deficiencies is dismissed as "flyspecking" because a close analysis of the EIS is necessary to understand the defendants' deception, this Court's commands that agencies disclose the worst case will become mere empty words.

A. The EIS Underestimates Exposure to the Pesticides and Consequent Risks to Health.

The EIS underestimates levels of exposure to, and therefore risk from, the pesticides it proposes to spray. Even for purposes of its "worst case" analysis, the EIS assumes that very little pesticide will be absorbed through the skin when unrebutted evidence shows that a significant amount will be absorbed. It assumes that pesticide residues will disappear from fruits, vegetables, animal meat, and water shortly after spraying, despite the fact that its own data show that the chemicals will persist for much longer periods of time. It assumes that most of the pesticides will be intercepted by trees and will not reach the ground, a clear error pointed out in this

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Court's previous opinion in this case. As a result of these assumptions, the EIS fails to present either the worst case consequences of spraying or even the most likely consequences.

1. The EIS does not use worst case skin absorption rates.

Absorption of pesticides into people's bodies is a serious impact that was thoroughly briefed and the subject of substantial testimony below. Absorption is one of the most critical factors in determining the "dose" that humans receive when sprayed with chemicals.[21] Unfortunately, the lower court's opinion does not contain any ruling on the adequacy of the EIS on this issue.[22] The EIS optimistically assumes that only 10 percent of the pesticides it proposes to spray can be absorbed through the skin. EIS at F-3. Yet the only available, published data on direct skin absorption of any of the four insecticides (Feldman & Maibach - 1974) (cited in EIS at F-29) show that in fact 73.9 percent of carbaryl is absorbed through the skin--more than seven times as much as defendants assume.

The EIS gives two excuses for not using the peer-reviewed and published study of Feldman and Maibach. First, the EIS claims that the 73.9 percent carbaryl absorption rate could have been affected by the acetone carrier used in conducting the experiment. EIS at F-29. As Dr. O'Brien testified, this assumption ignores the fact that the absorption rates of numerous other insecticides tested in the study with the acetone carrier were as low as 0.4 percent; this suggests that acetone as a "carrier" does not necessarily cause higher than normal absorption rates. ER 58; Statement of O'Brien at 3. No studies have ever shown acetone to allow an artificially high amount of carbaryl to be absorbed through the skin relative to other carriers. ER 101; Statement of Dr. Maibach at 2. In fact, no skin study has ever been published that suggests any absorption rate other than 73.9 percent. Dr. Maibach testified:

> At the moment there is no better data in man than was generated a decade ago [Maibach's study]. Unfortunately, in my opinion and in my judgment, that data has to stand until somebody is motivated in the public interest to repeat those studies using any vehicle [carrier] that is appropriate to be used. But in the meanwhile, since we have nothing better to go by, that is clearly the most relevant study that would be available.[23]

Defendants produced no data to support their assertion that an acetone carrier may have contributed to artificially high absorption rates.

The second excuse given by defendants for ignoring the 73.9

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[21] Exposure is the amount of a chemical with which one comes into contact. Dose is the amount of chemical which actually enters the body. The higher the absorption rate, the higher the resultant "dose" and the risk of consequent health effects. Defendants' use of an absorption rate only one seventh that indicated by the only published study greatly underestimates human health risks.

[22] Although one subheading of the opinion is entitled "Absorption and Persistence Rates," absorption rates are never discussed (the court refers once briefly to persistence and absorption of acephate on vegetables but completely ignores the serious issue of absorption through human skin).

[23] Dr. Maibach, one of the world's leading authorities on absorption of chemicals through the skin, was a member of the U.S. Environmental Protection Agency's science advisory panel for the registration of carbaryl. ER 168; RT at 192. Defendants' witness Dr. Dost (a veterinarian) testified: "I have to acknowledge that Dr. Maibach is an eminent authority in the field of percutaneous [through-the-skin] absorption." ER 165; RT at 438. Yet in this supposed worst case analysis, defendants chose to make optimistic assumptions about absorption through the skin rather than using unrebutted facts reported by a leading expert in the scientific literature. They assumed the best case, using an undocumented, low (10 percent) dermal absorption rate, and ignored the worst case (73.9 percent).

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percent rate is that "[i]f a 73.9 percent dermal absorption rate was used to calculate dosages, observers would receive . . . more than 5 times the highest dose actually recorded in mixer/loaders." EIS at F-30. In other words, defendants claim they have never measured doses of carbaryl in exposed people at levels that would prove a 73.9 percent absorption rate, and therefore high absorption rates could not occur. But the EIS deceives. The references it cites did not "actually record" dosage at all. Rather, dosage was estimated in two unpublished, non-peer-reviewed field studies (Schultz et al., 1979; and SCESC 1978) using an indirect, unpublished, and unverified Union Carbide method of estimating carbaryl dosage by measuring a metabolite of carbaryl (1-napthol) in urine. EIS at F-28, F-29. ER 58, 59; RT at 193. O'Brien Statement at 3, 4.

Defendants' use of unpublished studies, based on unpublished, and unverified industry methodology, rather than a peer-reviewed and published study by one of the world's foremost experts violates the requirement of 40 C.F.R. § 1502.24 that agencies "insure the professional integrity . . . of the discussions and analyses in environmental impact statements."[24]

Even if the unpublished Union Carbide approach appeared to be valid, and defendants presented some proof that an acetone carrier results in artificially high absorption rates, defendants

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[24] If defendants disagree with Dr. Maibach's study, the proper solution is not to use completely unreviewed industry information, the professional integrity of which cannot be insured. Rather, defendants must verify which is correct by conducting additional studies. 40 C.F.R. § 1502.22(a); Save Our ecoSystems v. Clark, 757 F.2d at 1249. See part II of this brief, supra (discussing agency research obligations).

have no right to calculate risk in a worst case analysis based solely on the less-worrisome Union Carbide study. The 73.9 percent absorption rate found by Dr. Maibach is at least the worst case. As this Court stated in Southern Oregon Citizens Against Toxic Sprays v. Clark, "[t]he agency may not omit the analysis only because it believes that the worst case is unlikely." 720 F.2d at 1429.

This is an absolutely fundamental error. To illustrate its importance by just one example, defendants' witness Dr. Dost cited a study (Maitlen et al. 1982) in which worker/mixer exposure was calculated to be one hundred times higher than the "worst case" exposure estimate assumed in the EIS, and calculated that applying a 75 percent absorption rate under those circumstances would result in a dose of 16 milligrams per kilogram of body weight. CR 209; Statement of Dost at 6. This is 5 times higher than the 3.125 mg/kg/day threshold for birth defects. See EIS at F-116, Table 2.[25]

2. The EIS understates initial pesticide residues on food.

In determining risk from dietary exposure to defendants' pesticides, the EIS underestimates the initial toxic chemical residues that are deposited on food by spraying and overestimates

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[25] While the study that Dr. Dost cited was ignored by the EIS for worker/mixer exposure levels, it was relied upon by the EIS for observer exposure levels, which it reported as lower than some other studies (EIS at F-3, line 2). At trial, Dr. Dost testified that the Maitlen et al. study would indicate mixer/loader worst case exposure would be above the NOEL for birth defects even if only 10 percent were absorbed. RT at 463-464. In short, the EIS ignored the study when it showed high exposure levels and used it when it showed low levels. Once again the preparers of the EIS seem to have made every effort to minimize the appearance of risk.

the amount that is washed off. The district court did not apply the "hard look" at EIS's that is required, in that the court failed to address several of the defendants' most serious omissions and simply misunderstood the evidence for others.

a. The EIS distorts pesticide residues on vegetables and fruit.

The treatment of exposure to carbaryl through eating vegetables and fruits illustrates the EIS's internal inconsistencies and misuse of data. The EIS cites a Michigan study in which carbaryl was applied at 1 pound per acre (the rate used throughout the EIS and proposed for carbaryl use), and left residues on leaves of 500 parts per million (ppm) one day after spraying. EIS at 49. The EIS then states that "typical" carbaryl residues on forest foliage after spraying range"from 30 ppm to 100 ppm." Id. But the EIS drops residue levels for food much lower before calculations begin. When discussing exposure from eating fruits and vegetables, the EIS concludes that "initial insecticide residues could range from 10 to 50 ppm." EIS at F-43; ER 59; RT at 23-24; 29-30 (Statement of O'Brien at 4). What happened to "typical" residues of 30-100 ppm? Would not the worst case at least use the actually recorded concentration of 500 ppm? Why would food plants receive less spray than non-food plants? The lower court did not address this issue.

The EIS next cites a study in which washing "shortly after spraying" removed 90 percent of carbaryl residues. EIS at F-43. The EIS then assumes, without further data, that 90 percent of trichlorfon and diflubenzuron residues (of its contrived 10 - 50 ppm) can also be washed off and that humans will wash fruits and vegetables "shortly after spraying." EIS at F-43. But there are no studies cited in the EIS that suggest that any percent of diflubenzuron (trade name - Dimilin) or trichlorfon residues will be removed by washing. In fact Dr. Neisses testified that "[t]here is a little data that the triflubenzuron [sic] may be picked up into foliage or cottonseed if it is applied to soil. Now, I don't know how to apply that to us applying dimilin [diflubenzuron] to foliage. So I handled the dimlin [sic] the same way I handled carbaryl and trichlorfon." ER 171; RT at 424. In other words, in the absence of knowledge, the EIS assumed the best -- that the chemical would also be washed off, despite "a little" disturbing data that chemical sprayed on adjacent soil might be picked up by the roots and added to that already deposited directly on the fruit or vegetable. This use of a 90 percent wash off assumption after professing ignorance of any supporting data and in spite of contradicting data is impermissible, especially for a worst case analysis.

The EIS admits that only five percent of acephate will wash off of fruits and vegetables. EIS at F-43. After acknowledging that "only 5 percent acephate residues can be removed by washing" id. (leaving 95 percent on food), the EIS blithely continues: "Therefore, acephate residue levels would range from 4.0 to 11.4 ppm after washing." Id. But this is not 95 percent of defendants' assumed initial residues of 10-50 ppm; it is 40 percent to 22.8 percent of those residues. At trial, defendants' witness Dr. Neisses attempted to explain, post hoc, this discrepancy. Dr. Neisses claimed that he did not use the 10-50

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ppm initial residue levels used for the other three insecticides when dealing with acephate. He instead used acephate data from an unpublished, report by the manufacturer of Orthene, Chevron Chemical Co. (1973), showing residues of only 4.3 to 12.4 ppm three days after application. ER 170; RT at 423. He then extrapolated backwards to "get a value of about 12 at day one." Id. This raises two problems. First, the Chevron figure contradicts statements in the EIS that acephate residues should be the same as other pesticides.[26] Second, Dr. Neisses did not explain how he extrapolated back. There are no documents in the EIS showing any such extrapolation. Apparently, only Dr. Neisses understands the method -- if it exists at all.[27] This post hoc rationalization is not permissible. Burlington Truck Lines v. U.S., 371 U.S. 156, 168, 83 S.Ct. 239, 246 (1962).

The reason for such misuse of data in the EIS is transparent. Using a residue of 47.5 ppm acephate (5 percent

washed off the previously acknowledged 50 ppm) in the calculations would have meant that the scenarios for all worst case categories that include a dietary component would exceed the Acceptable Daily Intake (ADI) and the No-Observable-Effect-Level (NOEL). See ER 175; RT at 34-35 (testimony of Dr. O'Brien); EIS, Table 8 at F-123. Furthermore, if only 5 percent were washed off, even using the low 10 ppm residue, leaving 9.5 ppm, all the "realistic" expected doses would still exceed the ADI for acephate. Id. To avoid reporting these disturbing results, the EIS simply assumes 4-12 ppm initial acephate residues instead of the 10-50 ppm it uses for other chemicals (or the 30-100 ppm in animal food, see next section; or even 500 ppm). Again, the EIS rejects its own assumptions and data when the predetermined conclusion of "acceptable margin of safety" is jeopardized.

b. The EIS distorts pesticide residues on meat.

The EIS underestimates the amount of pesticides humans may receive by eating livestock and game animals. When it determines the amount of chemicals that will be directly sprayed on these animals, the EIS cites two forest studies to conclude that two-thirds of the aerially applied chemicals will be intercepted by trees. EIS at F-32. ER 61; Statement of O'Brien at 6. The assumption that two-thirds of the spray will be intercepted (and thus animals will be exposed to only one-third), however, is no more appropriate for pastures throughout the nation than it was for South Salem, Oregon. See OEC v. Kunzman, 714 F.2d 901, 904 (9th Cir. 1983)(may not assume interception by foliage when spraying in non-forested areas). Actual spray residues may therefore be three times higher than the EIS assumes.

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[26] In response to a public comment the EIS notes: "If acephate were applied at the same application rate, there is no reason not to expect that acephate would be deposited at the same rate per hour as carbaryl." EIS at G-8, response to comment h. The EIS further states: "[T]he assumption that one pound of any insecticide, applied in similar methods, will result in one pound of insecticide being deposited on an acre of ground independent of the insecticide seems logical." EIS at G-6, response to letter 12 comment c (emphasis added). This means that if initial carbaryl, trichlorfon and diflubenzuron residues are 10-50 ppm (although 500 ppm have been documented), initial acephate residues will be 10-50 ppm, not 4-12 ppm. In short, the EIS assumes that if any two chemicals are applied at the same rate, residues will be deposited at the same rate. But it then decides that acephate residues will not be deposited on fruits and vegetables at the same rate as the other three insecticides. This is flatly illogical and significantly understates health risks.

[27] The use of data that is unavailable to the general public violates 40 CFR § 1502.21. See California v. Block, 690 F.2d 753, 765 (9th Cir. 1982).

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The EIS uses goats as an example of a meat animal and extrapolates from goats to other animals. EIS at 40. The EIS assumes goats grazing in sprayed areas will eat foliage with residues of only 100 ppm in the worst case (EIS at F-36) even though 500 ppm has actually been found. EIS at 49. Plainly livestock (at least in the worst case) will be exposed to levels of pesticides significantly higher than those assumed by defendants. Humans would therefore be expected to ingest higher amounts of insecticides via meat.

3. The EIS underestimates the persistence of the chemical pesticides it proposes to spray.

Plaintiffs pointed out below that the EIS underestimates the persistence--and therefore the ingestion by humans and the resulting health risk--of all four chemicals in food plants and water. The district court's response was that defendants' witnesses at trial criticized the studies on persistence that had been pointed out by plaintiffs (many of which studies were cited but not used or distinguished in the EIS itself) as unrepresentative of gypsy moth program conditions. But this is clearly not what defendants testified, and is a legally insufficient holding in any event. The court in effect said that defendants were free to use whatever data they wished. This is not true under NEPA.

a. The EIS understates the persistence of chemical pesticides on food plants.

The EIS improperly assumes that pesticide residues will completely disappear from food plants within 14 days. EIS at F-43-44. This assumption is contradicted by studies cited in the EIS itself and by defendants' witnesses at trial.

(1) Diflubenzuron persists on food plants longer than 14 days.

The EIS's assumption of a mere 14-day persistence for all pesticides on food plants was contradicted for diflubenzuron by several studies presented by plaintiffs. The EIS cited no diflubenzuron study at all to support its contention that diflubenzuron residues would degrade to zero within 2 weeks. The district court incorrectly stated that studies showing that diflubenzuron persists much longer than 2 weeks "had been in controlled environments not representative of the actual environment." 614 F. Supp. at 662.[28] But in fact plaintiffs cited field studies as well as controlled environment studies showing that diflubenzuron persists much longer than 14 days on food plants. Dr. O'Brien cited Nigg, et al. (1985) which found a half life of up to 16 weeks for diflubenzuron applied to citrus trees in the field. ER 70; Statement of O'Brien at 15. This study was not criticized by the defendants' witnesses. In fact, defendants' witness, Dr. Dost, a paid government reviewer of the EIS, testified that he coauthored a comprehensive literature review of diflubenzuron which included Nigg, et al. (1985). ER 166; RT at 461.[29] This review concluded: "It appears that

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[28] The lower court also said that witnesses stated that the "studies" of diflubenzuron and acephate used excessively high amounts that killed soil microorgamisms. 614 F. Supp. at 662. But no witness contradicted plaintiffs' evidence on the persistence of diflubenzuron and acephate. The district court apparently assumed testimony on the persistence of carbaryl (given by Dr. Quattlebaum, a Union Carbide employee - see RT at 400) went to all of the chemicals. It did not.

[29] Toxicological Evaluation of Dimilin, Oregon State University, Oregon State University Extension Service Toxicology Information Program, Department of Agriculture, Chemistry, Frank N. Dost, D.V.M., Extension Specialist in Toxicology and Chemistry.

Dimilin [diflubenzuron] is very persistent on foliage, but does not actually remain in its entirety as implied by the greenhouse data. Our conservative conclusion is that it will have a half life of 1 to 4 months." ER 166; RT at 461 (quoting Dost, Toxicological Evaluation of Dimilin). This would imply a degradation to zero residues measured in months or years (half of each remaining amount each 1-4 months), rather than days.

When asked why, as a paid reviewer of the EIS, he did not correct the assumption that diflubenzuron degrades to zero within 2 weeks, Dr. Dost stated: "I did not see that it was inaccurate. I didn't quite pick it up." Id. He excused this by stating that "the EIS was reviewed before we were able to get the documentation [and] was done at the most generous term on a crash basis." ER 167; RT at 462. But a study indicating extended persistence of diflubenzuron on plants was specifically brought to the defendants' attention in a public comment letter on the draft EIS. EIS, Appendix G, Letter 12 at 8. Defendants did not respond to the comment. In any event, haste to implement a program is no excuse for omitting significant environmental data, nor is the relaxed standard of review used by the district court appropriate.[30] Contrary to the opinion of the lower court, no criticism of the peer-reviewed, published studies presented by plaintiffs and commenters was offered by the defendants. The EIS's failure to use valid available data renders the EIS inadequate.

(2) Acephate persists on food plants longer than 14 days.

The EIS also assumes that acephate residues on fruits and vegetables will degrade to zero within 14 days (EIS at F-43). But the EIS itself cites studies on acephate that contradict this, stating:

> "Generally, a 5 to 10 day half life has been noted in plants (Chevron 1973). Wilcox (1973) reported that after applications of up to 0.5 lb. per acre, residues in leaves and litter dropped below 0.02 ppm (the limit of detection) in 33 days." EIS at 43 (emphasis added).

(A half life means that 50 percent of the material is still present. In 10 more days, 25 percent will still be present, and so forth.) On the very page where the authors assume zero pesticide residues after 14 days (F-43, first full paragraph), a Chevron Chemical Co. report is cited (eight lines earlier) that found 33 percent to 46 percent of residues remaining at the fourteenth day. Id. Although this same report was used to justify assumptions of low initial acephate residues (see part IV.A.2.a. of this brief, supra), it is ignored when it suggests longer persistence than defendants care to admit. The EIS cites

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[30] The district court ultimately allows the defendants to ignore studies on the assumption that "the agency's choice of studies on which to rely is within its discretion. I am precluded from reviewing such decisions unless I find them to be arbitrary and capricious." The court plainly applied the wrong legal standard. In Lathan v. Brinegar, 506 F.2d 677, 693 (9th Cir, 1974), this Court stated that "because NEPA is essentially a procedural statute . . . [we] think that the courts will better perform their necessarily limited role in enforcing NEPA if they apply [5 USC § 706(2)(D) in reviewing [EISs] for compliance with NEPA than if they confine themselves within the straitjacket of § 706(2)(A)." (Emphasis added.) This standard of review was most recently reaffirmed in Northwest Indian Cemetery Protective Association v. Peterson, 764 F.2d 581, 587 (9th Cir. 1985)(petition for rehearing pending). Lathan's view of § 706(2)(D) is plainly that it involves a more intensive review of an agency's actions under NEPA than does §706(2)(A). The district court's failure to apply the correct standard was error.

no studies at all to support its assumption that acephate degrades to zero in 14 days. Again, the trial court's disposition of these problems was to mention testimony involving the persistence of carbaryl (not acephate) in water (not plants), and to apply 5 U.S.C. § 706(2)(A) rather than § 706(2)(D).

(3) Carbaryl persists on food plants longer than 14 days.

The EIS's assumption of zero carbaryl residues on plants after 14 days is also contradicted by published studies on carbaryl cited in the EIS itself. Fairchild (1970) found 26 percent of initial carbaryl residues remaining 15 days after spraying on maple trees and 27 percent to 58 percent remaining 25 days after treatment of mixed oak stands. EIS at 49. In another project, carbaryl residues were high enough to kill 63 to 77 percent of gypsy moth larvae 60 days after spraying. EIS at 49. These studies from actual projects were never contested or explained away by defendants. The lower court simply did not respond to plaintiffs' contentions that carbaryl persistence on food plants had been underestimated. ER at 59, 60; RT at 33. Statement of O'Brien at 4, 5. The use of optimistic assumptions rather than actual data violates NEPA, and at a minimum the defendants' obligation to prepare a worst case analysis. As with diflubenzuron and acephate, the agency's discretion to ignore studies that show a greater environmental problem is severely limited when the agency is required to reveal the worst case.

(4) Trichlorfon persists on food plants longer than 14 days.

The EIS cites a study showing 15 percent of trichlorfon residues remaining on plants 60 days after treatment in a section

on trichlorfon's fate in the environment (EIS at 65), but when calculating realistic and worst case doses (EIS at F-43) it relies on another study indicating "nondetectable" residues and therefore concludes that residues will degrade to zero in 14 days. Id. As with other chemicals, the EIS assumes only the best in its risk assessment.

b. The EIS contradicts itself and erroneously assumes that chemical residues degrade rapidly in water.

The EIS improperly assumes that residues of all four insecticides will vanish from water within 5 days. EIS at F-42. This assumption is flatly contradicted by data that the EIS itself cites. The lower court failed to address this issue in the section on persistence.

The only study cited in the EIS regarding acephate's persistence in water reports a half life of about 50 days, not 5 days. EIS at 43.[31] The EIS's assumption that carbaryl residues persist in water for only 5 days (EIS at F-42) is likewise a "best case" rather than a "worst case" analysis. Defendants admitted that a study reporting carbaryl persisting in ponds 14 months after application (Gibbs, et al. 1984) would have

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[31] In response to a public comment citing a study (Tucker and Stevens, 1978) reporting a half life of acephate in water of 16-66 days (EIS App. G, Comment Letter #12 at 5), the EIS authors state they could not obtain the seven year old study from EPA before finalizing the EIS. EIS at G-10, response m. But NEPA requires an agency to obtain from another agency data before going forward with projects. 40 C.F.R. §§ 1502.2(f), 1506.1. To allow an agency to get around this requirement simply because a study takes time to obtain is contrary to the action-forcing purposes of NEPA. At the very least, data reporting a half life of 50 days (Chevron 1975) or 16-66 days (Tucker and Stevens 1978) for acephate in water constitute the worst case and must be included in the analysis.

been included in the EIS "if it had been brought to our attention during the comment period." ER at 115; Statement of Neisses at 6-7. But "under NEPA, [the] agency, not plaintiff, is responsible for investigating . . . environmental effects . . . ." Thomas v. Peterson, 753 F.2d 754, 765 (9th Cir. 1985) citing City of Davis v. Coleman, 521 F.2d 661, 671 (9th Cir. 1975).

The EIS claims that diflubenzuron residues in water will degrade to zero in five days, but cites absolutely no data. This optimistic assumption in the face of (1) uncertainty and (2) evidence that other chemicals persist much longer, is not a worst case analysis.

The EIS fails to consider adequately, at least in the worst case, the persistence of its pesticides on plants. It also fails to address adequately the persistence of acephate, carbaryl, and diflubenzuron in water. Rather than apply valid studies (many of which the authors themselves cite), the EIS chooses the most optimistic studies (even in the worst case analysis) to lend support to its decision to spray. This violates 40 C.F.R. § 1502.22 and the defendants' obligations to consider cumulative impacts. The district court confused the chemicals and studies, and deferred to the government's discretion in a manner that is inappropriate under NEPA and in a worst case analysis.

B. The EIS Improperly Manipulates ADI's and NOEL's.

The EIS improperly manipulates the use of "safety margins" for human exposure to defendants' pesticides, and ignores and manipulates data to make it appear as though No-Observable-Effect-Levels (NOEL) and Acceptable Daily Intake (ADI) safety thresholds will not be exceeded, when defendants'

own data, if properly applied, reveal that both NOEL's and ADI's in fact will be exceeded. Consequently, the EIS does not present a fair view of risks or a proper worst case analysis.

1. The EIS does not use the lowest NOEL for carbaryl to predict safety margins

a. Omitted data, improper reliance on phone calls, and abdication

The most widely used chemical in gypsy moth spraying will be carbaryl. The lowest "No Observed Effects Level" (NOEL) cited in the EIS for carbaryl is 3.125 mg/kg/day for birth defects in dogs. EIS at F-116, Table 2. But the NOEL that the EIS used for carbaryl to establish "Acceptable Daily Intakes" (ADI's), which represent margins of safety for humans, is not this 3.125 mg/kg/day NOEL, but rather a three-times higher NOEL of 10 mg/kg/day. EIS at F-67. Even more disturbingly, the EIS paid no attention to data showing NOEL's not just three times, but 167 and 83 times lower than the 10 mg/kg/day figure. The lower court did not address these crucial deficiencies shown at trial.

A No Observed Effect Level is the highest dose of a chemical at which no adverse effect is observed in a given experiment and for a given effect. Chemicals are tested at different levels to determine the dose at which adverse effects appear. Several NOEL's may exist for any chemical that has been tested for different effects. An Acceptable Daily Intake (ADI) represents a margin of safety for humans. It is estimated from the lowest animal NOEL, generally using a safety factor of 100 (i.e. a factor of 10 to account for the possibility that humans are more sensitive than the test animal and an additional factor of 10 to account for normal human variation in sensitivity). See EIS at

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F-67. Sometimes a factor of only 10 is used if data are available for humans or if exceptionally comprehensive animal data are present. Sometimes a safety factor of 1,000 or more is used. CR 218; Statement of Thomas at 5. ADI's and NOEL's are the standards by which defendants purport to assess the non-genetic impacts of their chemicals on human health.

The critical factor in establishing Acceptable Daily Intake levels for humans is, as the EIS recognizes, that the lowest human or animal no-observed-effect level be used. EIS at F-67. As explained by defendants' witness Dr. Calabrese, a "regulatory agency accepts the value of the most sensitive animal model as the one from which any NOEL/ADI is adopted." ER 148; Statement of Calabrese at 6 (emphasis added).

Evidence was presented at trial that a NOEL much lower than defendants' 10 mg figure exists. The California Department of Health Services has established a carbaryl NOEL for human kidney function of .06 mg/kg/day, based on an actual human volunteer study. ER 93; Statement of Bendix at 4, ¶23. See also RT at 19 (testimony of Dr. O'Brien). As a result of defendants' omission of the lower California kidney function NOEL, decisionmakers and the public are never told that all of the worst case amounts of carbaryl exposure for the general public listed in the EIS's tables that include food (a dietary component) (for example, .124 mg for "direct and dietary" exposure of the general public) exceed the .06 mg/kg/day human kidney function NOEL. In other words, there is no safety margin at all and in fact effects on humans may be expected to occur in the worst case.[32] See EIS at F-124, table 9. This kidney function NOEL of .06 mg is 167 times lower than the 10 mg animal NOEL that the EIS used to establish ADI's. If the .06 mg NOEL had been used and an ADI established at either .006 mg (using a 10-fold margin of safety) or .0006 mg (using a 100-fold factor), all "realistic" and "worst case" doses involving food would exceed such an ADI.[33] See EIS at F-124, Table 9.

Defendants' witness, Dr. Dost, cited an effects level for carbaryl significantly lower than the 10 mg supposed "no-effects" level used to establish ADI's in the EIS. He stated that humans "experienced symptoms attributable to carbaryl" at .12 mg/kg/day. CR 209; Statement of Dost at 6. This dose, which is somewhat higher than the .06 mg/kg/day dose in the California NOEL, is still 83 times lower than the 10 mg/kg/day NOEL the EIS uses to establish ADI's. All worst case general public exposures that include a dietary component essentially equal or exceed this .12 mg/kg/day effects level (see the figures of .124, .1178, .174, and .116 mg in EIS at F-124, right side of table 9). Of course, an ADI calculated using this amount as a NOEL (actually, a no-effects level would have to be even lower, since effects

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[32] Furthermore, all realistic doses in table 9 that include a dietary component come within two to six times of this California .06 mg NOEL.

[33] Diflubenzuron and trichlorfon ADI's are properly determined in the EIS by decreasing lowest NOEL's by a safety factor of 100. EIS at F-22. Table 7. But the ADI used for carbaryl (0.1 mg/kg/day -- EIS at F-122) is only 30 times lower than the lowest NOEL that the EIS itself acknowledges for carbaryl (3.125 mg/kg/day (EIS at F-122)), and no ADI margin at all would exist if the EIS reported and used the California NOEL (.06 mg).

were observed at this level) would be significantly exceeded.

Yet the district court did not address at all whether defendants properly selected NOEL's in the first instance. The district court recognized at one point that "ADI's are typically 100 times lower than NOEL's" (614 F. Supp. at 663), but failed to address plaintiffs' unrebutted evidence that the carbaryl ADI was not based on the lowest NOEL cited in the EIS, and therefore was not 100 times lower than the true NOEL. RT at 40-41; testimony of Dr. O'Brien.

The 10 mg/kg/day NOEL is not cited in the summary of established NOEL's for carbaryl where a reader would expect to find it (EIS at F-115, Table 7). Defendants' entire basis for accepting the 10 mg/kg/day NOEL as legitimate is an unwritten "personal communication" with an employee of Union Carbide, the manufacturer of carbaryl. This "communication's" very existence is not verifiable by the reader. The use of this ADI and 10/mg NOEL violates 40 C.F.R. § 1502.21 which states: " No material may be incorporated by reference unless it is reasonably available for inspection by potentially interested persons within the time allowed for comment. Material based on proprietary data which is itself not available for review and comment shall not be incorporated by reference."[34]

The "personal communication" claimed to know how another agency, EPA, had come up with its ADI, and thus claimed to know

implicitly (only) what an appropriate NOEL would be. Even if the EIS could legally rely on this phone call to a chemical company, it cannot rely on an ADI standard or NOEL established by a different agency without defendants independently examining the data themselves. This violates the "non-abdication duty." OEC v. Kunzman, 714 F.2d 901, 905 (9th Cir. 1983).

The ADI for carbaryl is 100x lower than a NOEL, but the EIS never mentions that it is only 30 times lower than the lowest carbaryl NOEL cited by the EIS, and is above the NOEL established in California for human kidney function.

b. Immune suppression, NOEL's, and abdication

The EIS does not even address NOEL's for some important effects for which carbaryl has been tested. Although the immune system of rabbits is depressed by ingestion of 0.23 mg/kg/day of carbaryl (ER at 64; Statement of O'Brien at 9; RT at 428), the EIS ignored immunosuppression NOEL's simply because the EPA does not require them for pesticide registration. ER at 113-114; Statement of Neisses at 4, 5; RT at 428. But suppression of the immune system can have devastating effects (Acquired Immune Deficiency Syndrome, or AIDS, for example, is a disease in which the immune system is depressed). Defendants cannot ignore valid evidence of adverse health effects simply because EPA has not examined those effects. To do so violates the Ninth Circuit's ruling in this very case: "One agency cannot rely on another's examination of environmental effects under NEPA." OEC v. Kunzman, 714 F.2d 901, 905 (9th Cir. 1983). Despite plaintiffs' emphasis on the impropriety of defendants' abdication, the district court did not even address the issue.

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[34] See also SOS v. Clark, 747 F.2d 1240, 1248, n. 13 (9th Cir. 1984); California v. Block, 690 F.2d 753, 765 (9th Cir. 1982) (emphasis added). The defendants violate this regulation throughout the EIS by incorporating and using data unavailable to the public.

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2. The EIS does not use a consistent safety margin for acephate.

The ADI for acephate of .025 mg/kg/day (F-67; F-122), is only ten times lower than the lowest NOEL of .25 mg/kg/day. Id. No justification or explanation is offered for this low safety factor. If the standard reduction by a factor of 100 were used in establishing an ADI for acephate of .0025 mg/kg/day, Table 8 at F-123 would show that all general public exposures that include a dietary component would exceed such an ADI by 10 to 60 times. The EIS conveniently avoided disclosing this fact by using a margin of 10-fold instead of 100-fold for this one chemical. There is absolutely no reason given in the EIS for the non-uniformity of method. The only reason for decreasing the margin of safety for acephate was apparently to keep from revealing that the aerial application of acephate in populated areas might well be unsafe.

C. The EIS Improperly Uses LOAEL's in Place of NOEL's in Order to Hide Indications of Risk.

While the draft EIS consistently used NOEL's as the standard for estimating the margin of safety of calculated exposures, the final EIS twice abandons NOEL's and subtly switches to a higher standard, a "Lowest Observed Adverse Effect Level" (LOAEL). This disguises the worst case and hides it from view. The switches occur when projected doses would have been shown to exceed NOEL's for birth defects. The authors never mention that the NOEL's were exceeded and thereby hide from the reader the fact that certain calculated exposures may precipitate birth defects.

A LOAEL is the lowest level yet tested at which an adverse effect is observed. It is an unreliable standard for purposes of

safety because future testing may reveal that adverse effects are caused at doses above the NOEL, but lower than the LOAEL.[35] ER 65-66, Statement of O'Brien. While use of NOEL's as a threshold for effects may err on the side of safety, any error in using LOAEL's can result in danger.

The EIS uses LOAEL's when discussing the risk of birth defects for sensitive humans. The EIS claims to lower the carbaryl NOEL for birth defects 100 times, then states that the "worst case dose" for the general public (i.e., observer and dietary category) is eleven times below the reduced carbaryl NOEL. EIS at F-100. This is false. In fact, this dose (0.174 mg/kg/day; EIS at F-124, Table 9) exceeds the lowered birth defects NOEL (3.125 mg/kg/day lowered to .03125 mg/kg/day) by five times. A parenthesis following the sentence regarding the eleven times margin of safety "explains" that the margin of safety was based on a LOAEL. RT 50, O'Brien. (No carbaryl LOAEL is cited in the EIS that is eleven times higher than the 0.174 mg/kg/day exposure, however. EIS at F-115, Table 2.)

The same procedure is used for acephate, whose lowered birth defects NOEL (0.10 mg/kg/day from 10 mg/kg/day - Table 1, F-114) would have been seen to be exceeded by all worst case human exposures that include a dietary component. EIS at F-123, Table 8. The EIS claims that the worst case dose for the general public (i.e., observer and dietary category) is 15 times below

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[35] For example, while the carbaryl NOEL is 3.125 mg/kg/day, the LOAEL is 6.25 mg/kg/day. RT 56, O'Brien. Any dose between 3.125 and 6.25 mg/kg/day may eventually be shown to cause birth defects if and when an experiment is run using that dose. The LOAEL, 6.25 mg, is not a reliable guide to safety.

the reduced acephate NOEL, then slips in (in a parenthesis) that this "margin of safety" was in fact calculated on a LOAEL, not a NOEL. (No acephate LOAEL is cited in the EIS, however. EIS at F-114, Table 1.[36]) ER 66; Statement of O'Brien at 11; RT at 49-50.

When discussing risks, therefore, the substitutions of LOAEL's for NOEL's occur when the EIS's own data would have shown that NOEL's for birth defects are exceeded by calculated doses; no justification is given for the substitutions, nor is the reader capable of locating the LOAEL's.[37] This is not the "full and fair discussion of significant environmental impacts" that 40 C.F.R. § 1502.1 requires. The lower court did not rule on the manipulations involving LOAEL's.

D. The EIS Ignores Information Necessary to Determine Cancer Risk of Diflubenzeron.

In response to public comment, defendants admitted in their final EIS that 4-chloroaniline, a metabolite of diflubenzuron, may be a carcinogen. Rather than perform an analysis of the

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[36] While the authors claim to use LOAEL's here, they admit doing so only in parentheses that follow a sentence claiming worst case doses do not exceed lowered NOEL's (EIS at F-100, line 23). The sentence is flatly false and there is no discussion of why LOAEL's are suddenly and deceitfully used nor which LOAEL's are used. The obvious answer is that there is then no need to acknowledge that the NOEL for birth defects for sensitive persons is surpassed by EIS-calculated exposures. In fact, the EIS states that "it is important to note that the MOS's [margins of safety--in this case below the LOAEL] are still above the level of 10 which has traditionally been used to account for intraspecies "variability." (EIS at F-100). What is not stated is that margins of safety are traditionally applied to NOEL's, not LOAEL's, and that they are traditionally 100, not 10, to provide for interspecies and intraspecies variation.

[37] EIS switches from a NOEL to a LOAEL a third time when discussing worst case exposures of small animals and possible birth defects. See ER 65; Statement of O'Brien at 10.

implications of this, however, or take comments on it, defendants simply assumed without basis that the level of the risk from 4-chloroaniline will be no greater than the cancer risk from the other chemicals they examine. This violates NEPA.

1. The EIS assumes with no basis that the cancer risk of diflubenzuron will be less than acephate .

The EIS does not make worst case assumptions about the cancer potency of 4-chloroaniline, a byproduct of diflubenzuron. Defendants state that a National Cancer Institute (NCI) study, while insufficient to establish the carcinogenicity of 4-chloroaniline reveals that "uncertainty still exists about the carcinogenicity of 4-chloroaniline." In other words, it may well cause cancer, because the tumors that appeared in the test animals, though few, were of a rare type of cancer. EIS at F-21. In the "Evaluation of Risk" section defendants again emphasize uncertainty about the carcinogenicity of 4-chloroaniline but inexplicably conclude, "Since NCI found no conclusive evidence of the carcinogenicity of 4-chloroaniline, its cancer potency would certainly be less than . . . acephate['s]." But defendants' own witness Neisses testified that the cancer potency of 4-chloroaniline may in fact be 40 percent higher than acephate's (or, perhaps, lower). ER 125; Statement of Neisses at 16. EIS at F-83 (emphasis added). Defendants go from uncertainty about cancer to certainty about level of cancer risk in one paragraph with no explanation.

Defendants' conclusion that 4-chloroaniline is less carcinogenic than acephate is not supported by any discussion, calculation, or data. ER 70; Statement of O'Brien at 15. The

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lower court did not address this issue. NEPA requires that "environmental impact statements . . . shall be supported by evidence that the agencies have made the necessary environmental analyses." 40 C.F.R. § 1500.2(b) (emphasis added). The NEPA "analyses" requirement demands that when defendants assert that 4-chloroaniline's cancer potential would "certainly be less than . . . acephate['s]," they explain the reasoning behind that conclusion, and, if not convincing, assume instead the worst case. Without such an explanation, neither the courts nor the public can meaningfully comment on the accuracy of the conclusion. See 40 C.F.R. § 1500.1(b) ("scientific analysis . . . and public scrutiny are essential to implementing NEPA"). The discussion of the cancer potency of 4-chloroaniline is inadequate and violates NEPA.

2. The EIS failed to disclose important routes of exposure to diflubenzuron.

Although a 1979 EPA Decision Document states that diflubenzuron metabolizes to 4-chloroaniline in soil, water, and (food) plants as well as in animals,[38] defendants formulated an "exposure" estimate for 4-chloroaniline based only on exposure through eating meat. EIS at F-83, F-84. ER 70; Statement of Dr. O'Brien at 15. There can be no doubt that defendants knew of the additional routes of human exposure because the 1979 Decision Document revealing those additional routes of exposure is the same document that defendants cite for their fish exposure estimate. EIS at F-83. A proper worst case scenario would consider exposure to 4-chloroaniline through the soil, water, plants, and animals.[35]

The lower court did not respond to plaintiffs' contentions that the 4-chloroaniline cancer risk estimate is seriously flawed because it ignores evidence that human exposure may occur via water and food plants and because it does not use any stated risk calculation methodology. (ER 125; Statement of O'Brien at 15;

E. Defendants ignore relevant data on drift.

Defendants assume the maximum aerial drift of pesticides after spraying will be one mile. EIS at F-31. However, plaintiffs submitted unrebutted evidence that pesticides routinely drift up to 50 miles. ER 108; Affidavit of Dr. Zemansky at 4. Dr. Zemansky also points out that one of defendants' witnesses, Dr. Dost, testified at trial in Save Our ecoSystems v. Watt, 747 F.2d 1240 (9th Cir. 1983), "that drift from the spraying of herbicides in eastern Oregon also reaches crops in the Yakima Valley of Washington," a distance of well over 50 miles. ER 108; Affidavit of Dr. Zemansky at 4. Once again defendants' estimates on exposure are far from worst case. The district court did not address plaintiffs' evidence.

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[38] Diflubenzuron Decision Document, Environmental Protection Agency, Special Review Division, March 26, 1979. Washington, D.C. 106p (referred to by defendants' witness Neisses); ER 125; Statement of Neisses at 16.

[35] Even the assumptions about fish are optimistic and contrary to data. Fish is considered by the EIS as the worst case meat scenario for diflubenzuron (EIS F-83), but a bioconcentration factor of only 1.0 for diflubenzuron in fish is used (F-39) despite a study cited in public comments indicating diflubenzuron accumulates in fish 13 times. The government offered no response to this comment (EIS, Appendix G, Letter 12, p. 8). The 1979 EPA Decision Document employs a bioconcentration factor of 50 based on several studies.

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V. DEFENDANTS HAVE FAILED TO DISCLOSE THE TRUE RISKS FOR CHILDREN AND CHEMICALLY SENSITIVE INDIVIDUALS.

The district court erred in holding that the EIS adequately discusses safety margins for children and chemically sensitive individuals. The EIS, while professing particular concern for children and chemically sensitive individuals, applies the very same margin of safety for children and chemically sensitive individuals as it applies for average adults.

As discussed earlier in this brief (at part IV.B.1) and as stated by the district court (OEC v. Kunzman, 614 F. Supp. at 663), an Acceptable Daily Intake level (ADI) of a chemical is established by lowering the No Observed Effect Level (NOEL) by a "safety factor" of 100: a factor of 10 to account for the possibility that humans as a species are more sensitive than the test animal and an additional factor of 10 to account for normal human variation in sensitivity. This standard formula is used to establish the maximum level of exposure deemed acceptable for the maintenance of human health for normal persons.[36]

Defendants admit, however, that variation from least sensitive humans to highly sensitive humans can be up to 100-fold or higher, rather than the 10-fold range assumed for average individuals. EIS at F-99. Consequently, in order to satisfy the worst case requirements for children and chemically sensitive persons, defendants would have to reflect the 10-fold animal-to-human variance and a 100-fold human-to-human variance (this would establish a safety factor of 1000x below the NOEL for children and chemically sensitive individuals). But the defendants failed to do this when reporting supposed safety margins for sensitive persons. However, they fooled the lower court into thinking that they had accounted for sensitive persons. Untangling the deception requires some explanation.

On F-99 of the EIS, defendants state:

> in order to account for possible impacts to sensitive individuals, the NOEL's were reduced by an arbitrary safety factor of 100.... Although 100 is arbitrary it is based on a review of selected literature on variable human responses to foreign chemicals.... Depending on the specific substance..., there was a 3.7 - 100 fold variation in response. (Emphasis added).

The material quoted plainly states that human responses to chemicals can vary by up to 100-fold,[37] but it also states that the total "safety factor" used by defendants is only 100-fold. What happened to the 10x factor that is used to account for animal-to-human variability?

The district court cited this paragraph and concluded: "there was no evidence or testimony that sensitive individuals exceed a 100-fold factor. Accordingly, the FEIS adequately discussed the potential risks to chemically sensitive persons,

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[36] For example, if the NOEL were 520 mg/kg/day, the 10x human-to-animal variance factor would reduce it to 52 mg/kg/day. The normal human-to-human safety factor would then reduce it by an additional factor of 10, to 5.2 mg/kg/day, which would be the ADI for normal humans.

[37] This conclusion is supported by defendants' own witness, Dr. Calabrese, who stated that variance in sensitivity among individuals may not be totally accounted for even if the safety factors were 100 to 1000. ER 150; Statement of Dr. Calabrese at 8. See also, Calabrese, High Risks Groups for Acephate, Carbaryl, Diflubenzuron, and Trichlorfon wherein defendants' witness Dr. Calabrese wrote last year that "substantial evidence exists which indicates that human variation in the metabolism of various xenobiotics grossly exceeds a factor of 10 . . . the range may approach and at times exceed a factor of 1000-fold."

and plaintiffs fail in this contention." OEC v. Kunzman, 614 F. Supp. at 662. That, however, misses the point. The point is that defendants in the above paragraph made it seem like they were developing a safety factor for chemically sensitive humans, when in fact they accounted only for being chemically sensitive (100x) while failing to account for being human (i.e., the animal-to-human 10x factor was eliminated for this group of humans only). Later in its opinion the district court acknowledges that the 10-fold animal-to-human variation is completely separate from whatever human-to-human "safety factor" (whether 10-fold or 100-fold) is used: "ADI's are established by dividing NOEL's by a factor of ten to account for increased sensitivity of humans over the test animals used to establish the NOEL's. These figures are then reduced another ten times to account for more sensitive humans." OEC v. Kunzman, 614 F. Supp at 663 (emphasis added).

The result is that a total safety factor of 100x is used for average humans (10x for animals-to-humans and 10x among humans) and a total safety factor of 100x is also used for children and chemically sensitive individuals (no factor for animals-to-humans and 100x among humans). This use of an identical safety margin for both fails to account for the extra sensitivity in response to toxic chemicals from persons in the latter group. The district court did not recognize defendants' deception.[38]

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[38] This Circuit specifically criticized the previous EIS (not even on a "worst case" basis) because it did not discuss "the effects of . . . contact by children or those sensitive to chemicals." OEC v. Kunzman, 714 F.2d at 904.

CONCLUSION

The EIS cannot be read and understood by the general public, it is not based on adequate research, it fails to reveal cumulative impacts, it misuses and ignores critical data, it fails to make worst case assumptions, and it does not disclose what defendants' aerial application of toxic chemicals over populated areas will really do to children and chemically sensitive individuals. As this Court has ruled, courts will not "engage in chronic fault-finding." But by the same token, the courts will not tolerate the mere appearance of disclosure when the reality is the consistent downplaying of environmental effects — particularly in what is supposedly a worst case analysis.

This is the first purported worst case analysis to come before this Court for review of adequacy since the decision in Save Our ecoSystems v. Clark was handed down. The ruling on the adequacy of this document will set the standard for all worst case analyses to follow. The defendants' EIS on its nationwide, long-term gypsy moth program does not comply with NEPA or with the regulations of the CEQ. Appellants request that the district

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court's opinion be reversed to the extent that it found the EIS adequate in the above areas, and that the case be remanded for entry of an appropriate order.

Respectfully submitted,

Michael Axline

John E. Bonine

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Counsel for Appellants

ON THE BRIEF:
Michael McShane
Kerry Rydberg
John Starkey
Legal Interns

DATED this 26th day of November, 1985

STATEMENT OF RELATED CASES

There are no cases related to the instant case.

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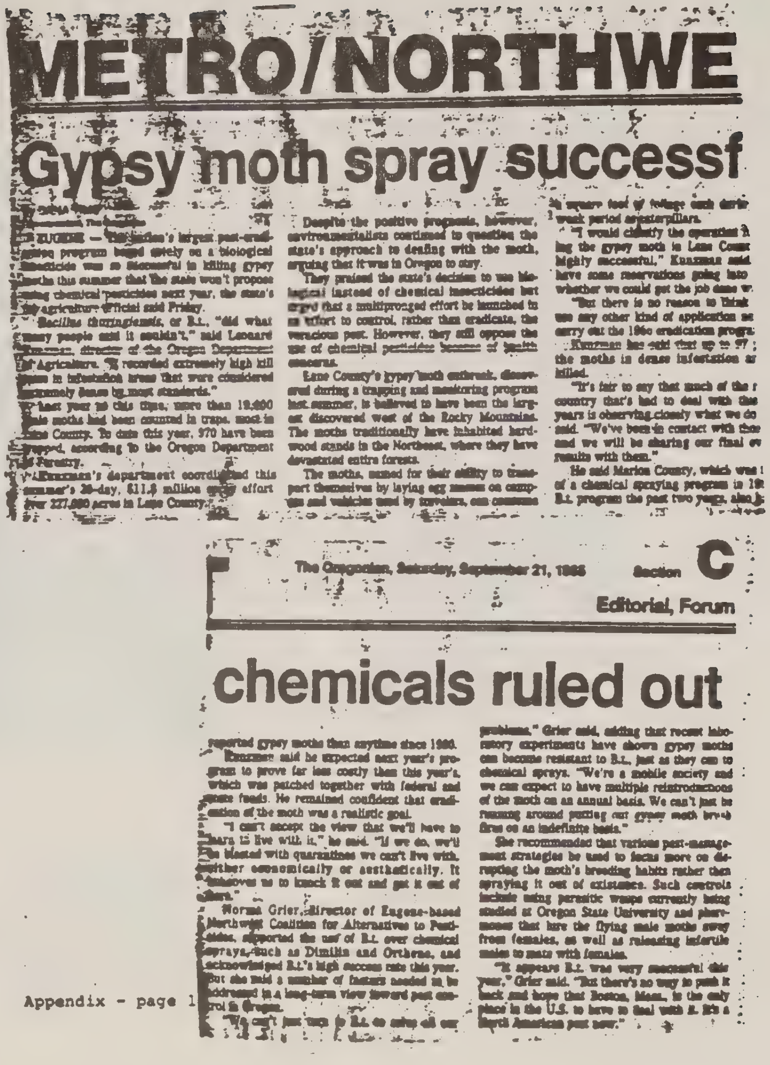

METRO/NORTHWE

Gypsy moth spray successf

EUGENE — The nation's largest pest-eradication program based solely on a biological insecticide was so successful in killing gypsy moths this summer that the state won't propose using chemical pesticides next year, the state's top agriculture official said Friday.

Bacillus thuringiensis, or B.t., "did what many people said it couldn't," said Leonard Kunzman, director of the Oregon Department of Agriculture. "It recorded extremely high kill rates in infestation areas that were considered extremely dense by most standards."

Last year at this time, more than 19,000 male moths had been counted in traps, most in Lane County. To date this year, 970 have been trapped, according to the Oregon Department of Forestry.

Kunzman's department coordinated this summer's 28-day, $11.8 million spray effort over 227,000 acres in Lane County.

Despite the positive prognosis, however, environmentalists continued to question the state's approach to dealing with the moth, arguing that it was in Oregon to stay.

They praised the state's decision to use biological instead of chemical insecticides but urged that a multipronged effort be launched in an effort to control, rather than eradicate, the voracious pest. However, they still oppose the use of chemical pesticides because of health concerns.

Lane County's gypsy moth outbreak, discovered during a trapping and monitoring program last summer, is believed to have been the largest discovered west of the Rocky Mountains. The moths traditionally have inhabited hardwood stands in the Northeast, where they have devastated entire forests.

The moths, named for their ability to transport themselves by laying egg masses on campers and vehicles used by travelers, can consume a square foot of foliage each durin week period as caterpillars.

"I would classify the operation ing the gypsy moth in Lane Coun highly successful," Kunzman said have some reservations going into whether we could get the job done w

"But there is no reason to think use any other kind of application carry out the 1986 eradication progra

Kunzman has said that up to 97 the moths in dense infestation a killed.

"It's fair to say that much of the country that's had to deal with the years is observing closely what we do said. "We've been in contact with the and we will be sharing our final e results with them."

He said Marion County, which was of a chemical spraying program in 19 B.t. program the past two years, also

The Oregonian, Saturday, September 21, 1985 Section **C**

Editorial, Forum

chemicals ruled out

reported gypsy moths than anytime since 1980.

Kunzman said he expected next year's program to prove far less costly than this year's, which was patched together with federal and private funds. He remained confident that eradication of the moth was a realistic goal.

"I can't accept the view that we'll have to learn to live with it," he said. "If we do, we'll be blasted with quarantines we can't live with, neither economically or aesthetically. It behooves us to knock it out and get it out of there."

Norma Grier, director of Eugene-based Northwest Coalition for Alternatives to Pesticides, supported the use of B.t. over chemical sprays, such as Dimilin and Orthene, and acknowledged B.t.'s high success rate this year. But she said a number of factors needed to be addressed in a long-term view toward pest control in Oregon.

"We can't just turn to B.t. to solve all our problems," Grier said, adding that recent laboratory experiments have shown gypsy moths can become resistant to B.t., just as they can to chemical sprays. "We're a mobile society and we can expect to have multiple reintroductions of the moth on an annual basis. We can't just be running around putting out gypsy moth brush fires on an indefinite basis."

She recommended that various pest-management strategies be used to focus more on disrupting the moth's breeding habits rather than spraying it out of existence. Such controls include using parasitic wasps currently being studied at Oregon State University and pheromones that lure the flying male moths away from females, as well as releasing infertile males to mate with females.

"It appears B.t. was very successful this year," Grier said. "But there's no way to push it back and hope that Boston, Mass., is the only place in the U.S. to have to deal with it. It's a North American pest now."

Appendix - page 1

APPENDIX

Letter 16
(continued)

ADDENDUM

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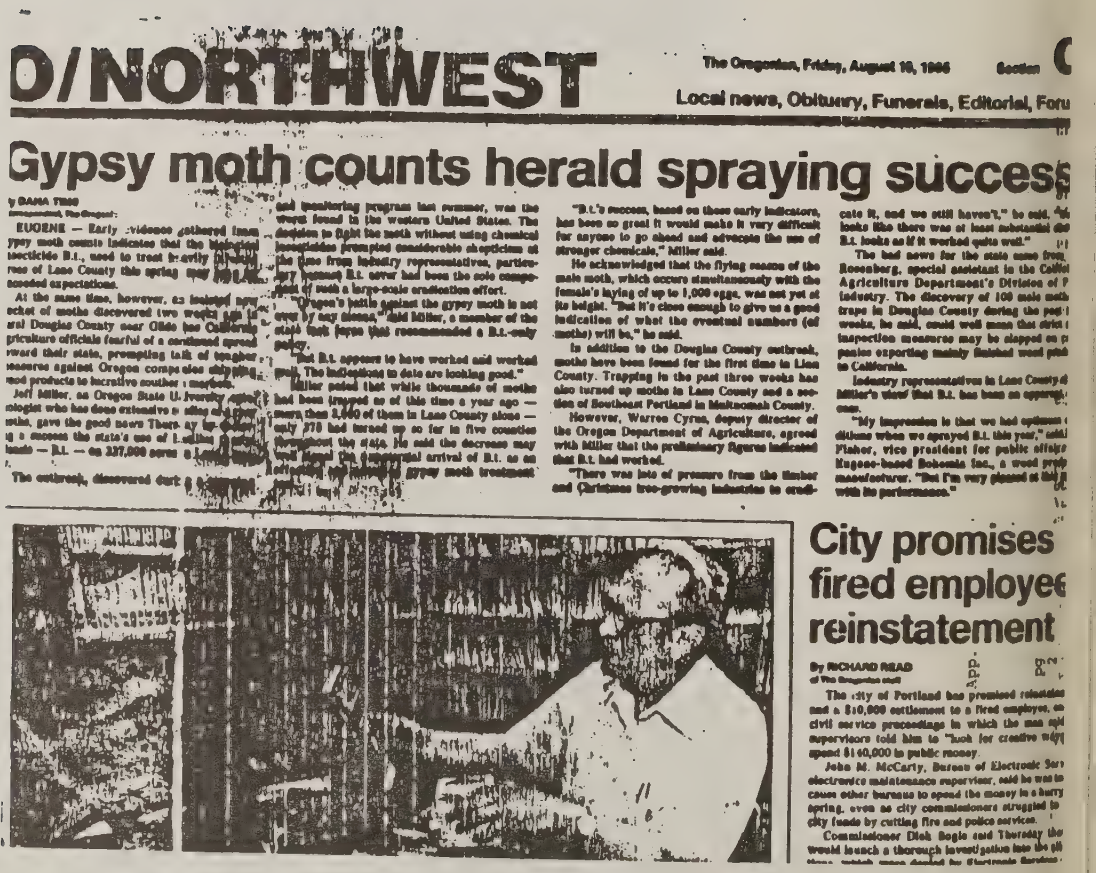

D/NORTHWEST

The Oregonian, Friday, August 15, 1986 Section C

Local news, Obituary, Funerals, Editorial, Foru

Gypsy moth counts herald spraying success

y DANA TIMS
[illegible], The Oregonian

EUGENE — Early evidence gathered from ypsy moth counts indicates that the biological nsecticide B.t., used to treat heavily [illegible] reas of Lane County this spring may [illegible] xceeded expectations.

At the same time, however, an isolated new ocket of moths discovered two weeks ago in ral Douglas County near Glide has California griculture officials fearful of a continued spread oward their state, prompting talk of tougher easures against Oregon companies shipping ood products to lucrative southern markets.

Jeff Miller, an Oregon State University entoologist who has done extensive studies of gypsy oths, gave the good news Thursday by declarg a success the state's use of [illegible] — B.t. — on 227,000 acres [illegible]

The outbreak, discovered during a [illegible] and monitoring program last summer, was the worst found in the western United States. The decision to fight the moth without using chemical insecticides prompted considerable skepticism at the time from industry representatives, particularly because B.t. never had been the sole component of such a large-scale eradication effort.

"Oregon's battle against the gypsy moth is not over by any means," said Miller, a member of the state task force that recommended a B.t.-only policy.

"But B.t. appears to have worked and worked well. The indications to date are looking good."

Miller noted that while thousands of moths had been trapped as of this time a year ago — more than 3,000 of them in Lane County alone — only 278 had turned up so far in five counties throughout the state. He said the decrease may well signal the [illegible] arrival of B.t. as an effective [illegible] gypsy moth treatment [illegible]

"B.t.'s success, based on these early indicators, has been so great it would make it very difficult for anyone to go ahead and advocate the use of stronger chemicals," Miller said.

He acknowledged that the flying season of the male moth, which occurs simultaneously with the female's laying of up to 1,000 eggs, was not yet at its height. "But it's close enough to give us a good indication of what the eventual numbers (of moths) will be," he said.

In addition to the Douglas County outbreak, moths have been found for the first time in Linn County. Trapping in the past three weeks has also turned up moths in Lane County and a section of Southeast Portland in Multnomah County.

However, Warren Cyrus, deputy director of the Oregon Department of Agriculture, agreed with Miller that the preliminary figures indicated that B.t. had worked.

"There was lots of pressure from the timber and Christmas tree-growing industries to eradicate it, and we still haven't," he said. "[illegible] looks like there was at least substantial [illegible] B.t. looks as if it worked quite well."

The bad news for the state came from Rosenberg, special assistant in the Califo Agriculture Department's Division of P Industry. The discovery of 100 male moth traps in Douglas County during the past weeks, he said, could well mean that strict inspection measures may be slapped on panies exporting mainly finished wood prod to California.

Industry representatives in Lane County Miller's view that B.t. has been an apparent cess.

"My impression is that we had optimum ditions when we sprayed B.t. this year," said Fisher, vice president for public affairs Eugene-based Bohemia Inc., a wood prod manufacturer. "But I'm very pleased at the with its performance."

City promises fired employee reinstatement

App. Pg 2

By RICHARD READ
of The Oregonian staff

The city of Portland has promised reinstate and a $10,000 settlement to a fired employee, a civil service proceedings in which the man said supervisors told him to "look for creative way spend $140,000 in public money.

John M. McCarty, Bureau of Electronic Ser electronics maintenance supervisor, said he was to cause other bureaus to spend the money in a hurry spring, even as city commissioners struggled to city funds by cutting fire and police services.

Commissioner Dick Bogle said Thursday the would launch a thorough investigation into the al [illegible]

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CEQ Regulations - 40 C.F.R. sections

§ 1500.1 Purpose.

(a) The National Environmental Policy Act (NEPA) is our basic national charter for protection of the environment. It establishes policy, sets goals (section 101), and provides means (section 102) for carrying out the policy. Section 102(2) contains "action-forcing" provisions to make sure that federal agencies act according to the letter and spirit of the Act. The regulations that follow implement section 102(2). Their purpose is to tell federal agencies what they must do to comply with the procedures and achieve the goals of the Act. The President, the federal agencies, and the courts share responsibility for enforcing the Act so as to achieve the substantive requirements of section 101.

(b) NEPA procedures must insure that environmental information is available to public officials and citizens before decisions are made and before actions are taken. The information must be of high quality. Accurate scientific analysis, expert agency comments, and public scrutiny are essential to implementing NEPA. Most important, NEPA documents must concentrate on the issues that are truly significant to the action in question, rather than amassing needless detail.

(c) Ultimately, of course, it is not better documents but better decisions that count. NEPA's purpose is not to generate paperwork—even excellent paperwork—but to foster excellent action. The NEPA process is intended to help public officials make decisions that are based on understanding of environmental consequences, and take actions that protect, restore, and enhance the environment. These regulations provide the direction to achieve this purpose.

§ 1500.2 Policy.

Federal agencies shall to the fullest extent possible:

(a) Interpret and administer the policies, regulations, and public laws of the United States in accordance with the policies set forth in the Act and in these regulations.

(b) Implement procedures to make the NEPA process more useful to decisionmakers and the public; to reduce paperwork and the accumulation of extraneous background data; and to emphasize real environmental issues and alternatives. Environmental impact statements shall be concise, clear, and to the point, and shall be supported by evidence that agencies have made the necessary environmental analyses.

§ 1502.1 Purpose.

The primary purpose of an environmental impact statement is to serve as an action-forcing device to insure that the policies and goals defined in the Act are infused into the ongoing programs and actions of the Federal Government. It shall provide full and fair discussion of significant environmental impacts and shall inform decisionmakers and the public of the reasonable alternatives which would avoid or minimize adverse impacts or enhance the quality of the human environment. Agencies shall focus on significant environmental issues and alternatives and shall reduce paperwork and the accumulation of extraneous background data. Statements shall be concise, clear, and to the point, and shall be supported by evidence that the agency has made the necessary environmental analyses. An environmental impact statement is more than a disclosure document. It shall be used by Federal officials in conjunction with other relevant material to plan actions and make decisions.

§ 1502.2 Implementation.

To achieve the purposes set forth in § 1502.1 agencies shall prepare environmental impact statements in the following manner:

(a) Environmental impact statements shall be analytic rather than encyclopedic.

(b) Impacts shall be discussed in proportion to their significance. There shall be only brief discussion of other than significant issues. As in a finding of no significant impact, there should be only enough discussion to show why more study is not warranted.

(c) Environmental impact statements shall be kept concise and shall be no longer than absolutely necessary to comply with NEPA and with these regulations. Length should vary first with potential environmental problems and then with project size.

(d) Environmental impact statements shall state how alternatives considered in it and decisions based on it will or will not achieve the requirements of sections 101 and 102(1) of the Act and other environmental laws and policies.

(e) The range of alternatives discussed in environmental impact statements shall encompass those to be considered by the ultimate agency decisionmaker.

(f) Agencies shall not commit resources prejudicing selection of alternatives before making a final decision (§ 1506.1).

(g) Environmental impact statements shall serve as the means of assessing the environmental impact of proposed agency actions, rather than justifying decisions already made.

40 C.F.R. sections

§ 1502.8 Writing.

Environmental impact statements shall be written in plain language and may use appropriate graphics so that decisionmakers and the public can readily understand them. Agencies should employ writers of clear prose or editors to write, review, or edit statements which will be based upon the analysis and supporting data from the natural and social sciences and the environmental design arts.

§ 1502.22 Incomplete or unavailable information.

When an agency is evaluating significant adverse effects on the human environment in an environmental impact statement and there are gaps in relevant information or scientific uncertainty, the agency shall always make clear that such information is lacking or that uncertainty exists.

(a) If the information relevant to adverse impacts is essential to a reasoned choice among alternatives and is not known and the overall costs of obtaining it are not exorbitant, the agency shall include the information in the environmental impact statement.

(b) If (1) the information relevant to adverse impacts is essential to a reasoned choice among alternatives and is not known and the overall costs of obtaining it are exorbitant or (2) the information relevant to adverse impacts is important to the decision and the means to obtain it are not known (e.g., the means for obtaining it are beyond the state of the art) the agency shall weigh the need for the action against the risk and severity of possible adverse impacts were the action to proceed in the face of uncertainty. If the agency proceeds, it shall include a worst case analysis and an indication of the probability or improbability of its occurrence.

§ 1508.7 Cumulative impact.

"Cumulative impact" is the impact on the environment which results from the incremental impact of the action when added to other past, present, and reasonably foreseeable future actions regardless of what agency (Federal or non-Federal) or person undertakes such other actions. Cumulative impacts can result from individually minor but collectively significant actions taking place over a period of time.

§ 1508.25 Scope.

Scope consists of the range of actions, alternatives, and impacts to be considered in an environmental impact statement. The scope of an individual statement may depend on its relationships to other statements (§§ 1502.20 and 1508.28). To determine the scope of environmental impact statements, agencies shall consider 3 types of actions, 3 types of alternatives, and 3 types of impacts. They include:

(a) Actions (other than unconnected single actions) which may be:

(1) Connected actions, which means that they are closely related and therefore should be discussed in the same impact statement. Actions are connected if they:

(i) Automatically trigger other actions which may require environmental impact statements.

(ii) Cannot or will not proceed unless other actions are taken previously or simultaneously.

(iii) Are interdependent parts of a larger action and depend on the larger action for their justification.

(2) Cumulative actions, which when viewed with other proposed actions have cumulatively significant impacts and should therefore be discussed in the same impact statement.

(3) Similar actions, which when viewed with other reasonably foreseeable or proposed agency actions, have similarities that provide a basis for evaluating their environmental consequences together, such as common timing or geography. An agency may wish to analyze these actions in the same impact statement. It should do so when the best way to assess adequately the combined impacts of similar actions or reasonable alternatives to such actions is to treat them in a single impact statement.

(b) Alternatives, which include: (1) No action alternative.

(2) Other reasonable courses of actions.

(3) Mitigation measures (not in the proposed action).

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40 C.F.R. sections

§ 1508.27 Significantly.

"Significantly" as used in NEPA requires considerations of both context and intensity:

(a) *Context.* This means that the significance of an action must be analyzed in several contexts such as society as a whole (human, national), the affected region, the affected interests, and the locality. Significance varies with the setting of the proposed action. For instance, in the case of a site-specific action, significance would usually depend upon the effects in the locale rather than in the world as a whole. Both short- and long-term effects are relevant.

(b) *Intensity.* This refers to the severity of impact. Responsible officials must bear in mind that more than one agency may make decisions about partial aspects of a major action. The following should be considered in evaluating intensity:

(1) Impacts that may be both beneficial and adverse. A significant effect may exist even if the Federal agency believes that on balance the effect will be beneficial.

(2) The degree to which the proposed action affects public health or safety.

(3) Unique characteristics of the geographic area such as proximity to historic or cultural resources, park lands, prime farmlands, wetlands, wild and scenic rivers, or ecologically critical areas.

(4) The degree to which the effects on the quality of the human environment are likely to be highly controversial.

(5) The degree to which the possible effects on the human environment are highly uncertain or involve unique or unknown risks.

(6) The degree to which the action may establish a precedent for future actions with significant effects or represents a decision in principle about a future consideration.

(7) Whether the action is related to other actions with individually insignificant but cumulatively significant impacts. Significance exists if it is reasonable to anticipate a cumulatively significant impact on the environment. Significance cannot be avoided by terming an action temporary or by breaking it down into small component parts.

(8) The degree to which the action may adversely affect districts, sites, highways, structures, or objects listed in or eligible for listing in the National Register of Historic Places or may cause loss or destruction of significant scientific, cultural, or historical resources.

ATTACHMENT 2

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IN THE UNITED STATES DISTRICT COURT
FOR THE DISTRICT OF OREGON

OREGON ENVIRONMENTAL COUNCIL,
Plaintiffs,
and
FRIENDS OF THE EARTH, et al.,
Plaintiffs - Intervenors,
v.
LEONARD KUNZMAN, Director,
State of Oregon, Department
of Agriculture, et al.,
Defendants.

Civil No. 82-504-RE

NARRATIVE STATEMENT OF MARY
O'BRIEN IN BEHALF OF
PLAINTIFFS

I, Mary Hallie O'Brien, Ph.D., am a botanist currently employed as Information Coordinator and NCAP NEWS Editor by the Northwest Coalition for Alternatives to Pesticides (NCAP), a five-state coalition of citizen groups working for pesticide reform. My area of expertise is interpretation of scientific literature for lay readers (see attached resume, Appendix A).

I have been asked by the Oregon Environmental Council to address the assessment of risks in the Gypsy Moth Suppression and Eradication Projects: Final Environmental Impact Statement as Supplemented - 1985 (FEIS). As a staff person with NCAP, a coalition whose member groups and individuals have repeatedly contended that health risks need to be addressed pursuant to 40 CFR 1502.22, I commented in writing on the Draft Supplement to the Final Environmental Impact Statement during the public comment period (Appendix B). I indicated that the Draft Supplement did not enable decisionmakers to clearly compare each pesticide so that before using public money to spray the public and the environment, the decisionmaker would be able to say, "This pesticide has these efficacy benefits, these environmental drawbacks, and these human health drawbacks" (the latter being the focus of the Draft Supplement). Rather, the Draft Supplement served to assure the decisionmaker that health risks need not figure into a final choice as to whether or which pesticides should be used in any given gypsy moth project.

The types of deficiencies identified in those comments remain in the FEIS with the result that risk is consistently and deliberately misrepresented and downplayed and the public and decisionmakers are deprived of the tools needed to make informed decisions about the gypsy moth insecticides. There are too many cases of scientific inaccuracy, ignored information, and numerical juggling to describe in an affidavit of reasonable length.

The instances of inaccuracy are substantial by themselves, but when compounded, result in a risk analysis that improperly eliminates risk as a concern. I illustrate general types of deficiencies by focusing on the example of carbaryl in parts I-IV. I touch on other insecticides in part V. In part VI, the arrangement of information in the FEIS is shown to be obfuscatory and therefore counter-productive for NEPA purposes.

I. EXPOSURE TO THE INSECTICIDES IS SYSTEMATICALLY UNDERESTIMATED.

A) Dermal exposure: According to the FEIS, only one study of human dermal (skin) absorption for any of the four insecticides has been conducted, that of Feldman and Maibach (1974; FEIS F29) in which carbaryl applied to the skin of the forearm (the region of our body that absorbs insecticides the least, according to Feldman and Maibach), was absorbed at a rate of 73.9%. The FEIS authors dismiss the study by saying that carbaryl's absorption could have been affected by the acetone carrier (although the absorption rate of other insecticides, also tested by Feldman and Maibach with the acetone carrier, varied from 0.4% to 73.9%).

The FEIS authors also note that a 73.9% absorption rate for carbaryl would mean that the conclusions of three unpublished field studies of carbaryl exposure (Schulze et.al. 1979; SCESC 1978, 1979 in FEIS) might be wrong (see F29-F30). These unpublished studies used an indirect method of estimating exposure to carbaryl via urinary analysis for a carbaryl metabolite (1-naphthol), a method

based on Union Carbide data (see F28;SCESC 1978). These studies are used by the FEIS for estimating exposure of workers, observers, and residents to all four gypsy moth insecticides.

No study of direct dermal exposure has been conducted that contradicts the Feldman and Maibach study. The FEIS therefore wrongly ignores the documented 73.9% absorption rate for carbaryl, and therefore reduces the exposure scenarios for workers, observers, the general public, and all accidental scenarios.

B. Dietary Exposure - Vegetables: According to the FEIS, "typical" carbaryl residues on forest foliage range from 30-100 ppm when carbaryl is applied for gypsy moth control, with 16-20% remaining after two-three weeks (F49). Residues of 500 ppm were found on maple trees one day after one pound active ingredient/acre was applied (the rate proposed for gypsy moth control); 11% remained 35 days later (F49). The FEIS cites another study showing residues remaining high after 60 days and causing 11% larval mortality after 114 days (Doane and Schaefer 1971; FEIS p.49).

When the FEIS comes to food crops, however (i.e. human risk), initial residues are estimated (in the worst case analysis) to be 10 to 50 ppm, and are assumed to degrade to zero within 14 days (F43). Moreover, it is assumed that 90% of the carbaryl can be washed off, so initial residues are considered to be 1 to 5 ppm. The only data cited for this indicates initial residues of 52 ppm on spinach with no application rate mentioned (Kuhr and Dorough

1976 in FEIS F43).

The FEIS wrongly states that "...insecticides are usually rapidly degraded...or, if not degraded, translocated to often inedible plant parts" (F35). A pesticide safety factor report for the United States Department of Labor by Clement Associates (1979) notes that "because residues on the surfaces of plants on the soil surface dissipate more rapidly than those in the edible parts of the crops, crops may be safe to handle before they are safe to eat" (emphasis added). The Clements report recommends that children not reenter a field to harvest strawberries and potatoes until 40 days after carbaryl has been sprayed.

The importance of these considerations for the FEIS lies in its calculations of dietary exposure (initial residues) and carcinogenicity calculations (involving persistence of residues and consequent repeated ingestion of carbaryl).

C. Dietary Exposure - Water: The FEIS assumes that carbaryl residues will persist a maximum of five days. Three unpublished studies (one by Union Carbide) and one published study are cited for this assumption (FEIS, p.50).

A study not cited by the FEIS indicates that carbaryl may persist for months in some waters: LaFleur (1976) reports that "carbaryl residues were found in underlying ground water within two months after soil applications were made and persisted through the eighth month." Carbaryl did not disappear from a depth of

20 cm until after the fourth month and remained at a depth of 20-40 cm until the sixteenth month. This persistence of carbaryl in water would increase the calculated risk of carcinogenicity as well as pose potential groundwater and drinking water contamination problems not mentioned in the FEIS.

D. Dietary Exposure - Meat: Based on two forest studies (Maksymiuk and Orchard 1975; Newton and Dost 1981 in FEIS F32), the FEIS assumes that two-thirds of an insecticide application will be intercepted by foliage. Hence, the potential exposure to domestic and wild animals is estimated to be 35 mg/m^2, rather than the full 100 mg/m^2 applied (at one pound active ingredient/acre).

This assumption is not justified for cleared urban or rural land. Aside from underestimating hazards posed to nonhuman animals exposed to carbaryl by gypsy moth programs, the assumption of foliage interception of spray similarly results in an underestimation of the potential dietary exposure to humans who eat domestic animals.

Conclusion: By the time the FEIS assumes that (1) only 10% (rather than 73.9%) of carbaryl is absorbed through human skin, (2) only 1-5 ppm carbaryl (rather than 100 to 500 ppm) will be initially present on food, (3) carbaryl residues will remain in water 5 days (rather than months) and (4) two-thirds (rather than none) of the spray will be intercepted by trees, the outcomes of the FEIS calculations are seriously compromised.

II. EFFECTS OF LOW-LEVEL EXPOSURE ARE MISPRESENTED

A. The NOEL is Misrepresented: The NOEL is described in the FEIS as "an acceptable standard for evaluating overall human health risks because it is based on the evaluation and significance of the total data base (emphases added)." (F67). It is defined in the glossary as "the highest level (of dose) at which no effect is observed, i.e., safe in the species tested" (p.104). This is inaccurate:

1. A NOEL is technically a no-observed-adverse-effect level only for those tests that have been administered and for those effects studied. In fact, none of the four insecticides is unconditionally registered which means that none of them yet have all tests that will be required to be submitted and reviewed by EPA for full registration. So the data base cannot be considered "total".

2. Certain adverse effects are not included in the testing normally done for a NOEL and yet may pose a hazard that must be considered by decisionmakers. On p. 59 (but not in the worst case analysis), the FEIS mentions that carbaryl has been found to cause viral potentiation. The FEIS does not define what that means or explain the reason such an effect was studied. Reye's syndrome, a viral liver and brain disease frequently fatal in children, appears to have an environmental factor, is more prevalent in rural areas than in urban areas, and has been tentatively noted to increase among children following certain spruce budworm spray programs. Because certain pesticides and pesticide emulsifiers have been implicated as the possible environmental factor in the

development of Reye's syndrome, a study was undertaken to see if carbaryl, a pesticide widely used for the control of spruce budworm, would interact with varicella-zoster virus, the virus associated with Reye's syndrome in epidemiological studies. Carbaryl was found by the researchers (Abrahamsen and Jerkofsky 1981) to increase the virus 10-13 times over control cultures of virus-infected human embryonic lung cells.

Having explained none of this, the FEIS does mention that a panel of medical experts was convened by the Maine Bureau of Forestry to review these studies. The panel concluded that since carbaryl poses a "potential but inconclusive health risk...no uninformed or unconsented human exposure" should occur during a forest spray operation.

Having mentioned this viral enhancement problem, the FEIS concludes its review of carbaryl on the next page (FEIS, p.61) by saying that "...it is highly unlikely that the registered use of carbaryl, as applied to treatment areas during gypsy moth suppression or eradication projects would pose a human health hazard." How can this conclusion be made?

Tests for viral enhancement have not been included in determining any NOEL for carbaryl and the data base is therefore not "total", nor can the NOEL be regarded as the dose that is "safe" in the species tested.

B. The Implications of Approaching a NOEL are Misrepresented.

1. The FEIS notes in its conclusions on the toxicology of carbaryl that "the estimated worst case doses to (domestic and wild) animals are orders of magnitude below doses that cause effects in test animals, with the possible exception of teratogenicity in dogs (i.e. 6.25 mg/kg/day)" (FEIS p.53, emphasis added).

This is an unwarranted, inaccurate representation of NOELS. Following the administration of a single dose of 10 mg/kg carbaryl, rats learned much less quickly than the controls to avoid electric shock associated with pressing a lever for water (Sideroff and Santolucito 1972). This dose is only slightly higher than the 6.25 mg/kg/day dose that has been found to induce birth defects in dogs (FEIS p.53 and Table 2).

Similarly, the immune system of rabbits was depressed following administration of carbaryl in feed at rates as low as 0.23 mg/kg/day (Street and Sharma 1975). The authors note that the immune suppression appeared at levels which produced no tissue damage and that immunosuppression testing "is important in relation to health aspects of pesticides or other environmental chemicals, particularly the ones that are known to be persistent in the environment." (The paper notes that immune suppression has been found by other researchers studying trichlorfon, another of the FEIS gypsy moth insecticides.)

Because carbaryl testing data not normally included in

establishing NOELS (e.g. for learning effects and immune suppression), exists in the peer-reviewed scientific literature, the FEIS incorrectly compares human exposure risks only to their limited notion of NOELS (Tables 9 and 13).

2. When the FEIS estimated that the carbaryl NOEL for birth defects could be exceeded for small nonhuman animals following a gypsy moth spray program, the FEIS switched to a higher standard for judging the estimated exposure risk. The FEIS notes that the worst case dose estimate of carbaryl to small animals is 3.4 mg/kg/day (even with the supposed 2/3 interception of the spray by foliage, 10% dermal absorption, and 100 ppm rather than 500 ppm initial residues on foliage the animals might eat). Having noted on the previous page that the NOEL for birth defects in pregnant dogs is 3.25 mg/kg/day, the FEIS does not say "A pregnant dog exposed to carbaryl at the worst case dose might exceed the threshold for birth defects." Instead it says, "The teratogenic threshold for dogs is only 6.25 mg/kg/day (Table 2 in Appendix F) which is close to the estimated worst case dose to small animals of 3.4 mg/kg/day" (FEIS, p. 53). What happened to the NOEL of 3.25 mg/kg/day? The 6.25 mg/kg/day figure is not the NOEL but is rather the LOAEL, or Lowest Observable Adverse Effect Level, the lowest level tested that was shown to produce the effect studied. While the actual "lowest adverse effect" dose might be still lower (e.g. the estimated exposure of 3.4 mg/kg/day), if no-one has tested at that level, it would not be listed as a LOAEL. The LOAEL is a record of testing

that has been done, not a record of the lowest dose that may cause effects.

The LOAEL as used in the FEIS (it was not used in the Draft Supplement), is one more tool by which hazard is underestimated, downplayed, or denied. Surely a worst case analysis ought to use NOELS (i.e. the highest known dose at which certain effects are not observed), not LOAELS (i.e. the lowest dose so far tested that has produced negative effects).

3. Similarly, when the risk analysis estimates that certain worst case human exposure scenarios may exceed the margin of safety of 100X below the NOEL for birth defects (using data on dogs), higher NOELS are chosen from less sensitive species (mouse and rabbit) so that the 100X safety factor is restored (F94), "that exposures (even worst case) resulting from the use of any of the four insecticides to suppress or eradicate gypsy moth are below teratogenic thresholds and probably within margins of safety for the general population." Clearly the "worst case" is that exposures may not be within margins of safety, if the most conservative NOEL is used.

When discussing the risk of birth defects for sensitive populations, the higher LOAELS, not the NOELS, are used (F100). This is an apology for insecticides, not an objective examination of their potential health effects. It is not proper to create desired margins of safety by picking and choosing among various

NOELS, and LOAELS. A worst case analysis should rely on those documented studies that indicate damage at the lowest doses.

III. CUMULATIVE EFFECTS AND SYNERGISM ARE MISREPRESENTED

A. Carbaryl has been shown to increase the lethal toxicity of several other pesticides in trout (Statham and Lech 1975; FEIS at F102). According to the researchers, the mechanism may involve alteration of uptake of the other pesticides from water. In dismissing the study, the FEIS says that (1) since the concentrations in the study were higher than would be expected from the use of carbaryl for gypsy moth, the synergistic effects could be considered to be worst case. (However, that conclusion is never applied anywhere in the FEIS)(F102); (2) since the study involves effects in fish, the FEIS will instead estimate risk of carbaryl's synergism for humans only from the synergism of tobacco smoke with asbestos, ignoring the possibility that the carbaryl-pesticide synergism data might have relevance to humans; and (3) a 10-fold increase in toxicity of carbaryl would be the worst case (in which case, says the FEIS, the NOEL will not be exceeded), ignoring the fact that in the Statham and Lech study, it was toxic effects of the other pesticides, not carbaryl's, that were increased to a deadly level by the synergism.

B. A study not cited by the FEIS indicates that antidepressant drugs (monoamine oxidase inhibitors) could inhibit metabolism and excretion of carbaryl in animals, thereby allowing carbaryl to remain in its toxic form longer and to remain in the body longer (Culver, et al. 1970). The metabolism of carbaryl was reduced by as much as 71%. Those members of a population who are utilizing antidepressant drugs should be identified as a sensitive population of concern in any public spray program involving carbaryl. In my comments on the Draft Supplement, I pointed out the Statham and Lech synergism study to the FEIS writers. The writers, while including the study in the FEIS, basically discounted and dismissed it. Do citizens have to point out every such study to FEIS writers before it is included? What must a citizen do to have studies demonstrating "worst case" possibilities treated seriously in a "worst case analysis?"

C. By claiming that "to fully discuss cumulative effects, the total dose from all sources would need to be calculated" and so "can only be discussed in general terms," the FEIS dismisses the topic in three useless paragraphs (F103). At a minimum, the FEIS should discuss the implications of multiple applications of persistent gypsy moth insecticides. The finding that carbaryl residues remain "high" 60 days after spraying (FEIS p. 49) should alter the dietary exposure estimates and the risk analyses, given that most eradication programs utilizing carbaryl involve two or three applications during the course of two or three months (FEIS, Appendix E). No mention at all is made of the possible cumulative effects of multiple applications.

IV. CANCER RISK IS UNDERESTIMATED

As I noted in my comments on the Draft Supplement (see p.11, Appendix B), a study by William Lijinsky of the National Cancer Institute implies a much higher estimated cancer risk from N-nitrosacarbaryl, a metabolite of carbaryl, than is calculated in the FEIS. This issue is addressed in Dr. Lijinsky's written statement to the court and drastically alters the "worst case" scenario presented in the FEIS.

V. THE DEFICIENCIES ILLUSTRATED IN THE CARBARYL EXAMPLES (I-IV) ARE PERVASIVE THROUGHOUT THE FEIS

Lest my focus on carbaryl be construed to imply that the FEIS adequately addresses the risks of the other insecticides, I list several examples to the contrary:

1. My Draft Supplement comments compelled the FEIS writers to admit that their assumption that 90% of acephate residues will be washed off food is false. The FEIS states that "since acephate has a unique characteristic (of the four insecticides being analyzed) of being absorbed into or onto leaf surfaces....[t]his analysis assumed that only 5 percent of acephate residues would be removed through washing" (FEIS F43). The FEIS then proceeds on the assumption that 60-80% (not 5%) of the carbaryl residues will be removed, (FEIS, F43). This drastically affects the dietary exposure portion of the risk analysis.

2. As noted in my Draft Supplement comments, as much as 90% of intact diflubenzuron residues have been found to remain on

plants 30 to 60 days after spraying. A greenhouse study indicates that 89 to 100% of diflubenzuron residues remain on soybean, apple, corn, and cabbage leaves 8 weeks after spraying (Verloop and Ferrell 1977). Applied to citrus trees, half of the diflubenzuron residues remained as long as 16 weeks later on the fruit (Nigg et al. 1985). Despite this, the FEIS contends that residues of diflubenzuron will disappear from food plants within 14 days. (FEIS F43). This unsupported optimistic conclusion is another example of how the FEIS develops "best case" scenarios for the impact of the gypsy moth insecticides on human health.

3. Despite the summary table that confidently assures decisionmakers that the risk of contracting cancer from any of the four insecticides is in the "1 in a million range" (FEIS, p. 17), the processes used to arrive at that assurance are at best questionable:

a. Diflubenzuron: On F83, the reader learns that the diflubenzuron cancer risk calculation did not use the cancer potency model or risk assessment used for the other three insecticides. Moreover, the cancer risk caluclation is not even based on diflubenzuron or 4-chloroanalinecancer experimental data. Rather, the FEIS (1) wrongly assumes a person will encounter 4-chloroanaline only in meat; (2) considers that meat consumption makes up only 8% of the total exposure to diflubenzuron; and (3) notes that less diflubenzuron is used per acre than the other three insecticides. The wrong conclusion is that exposure to diflubenzuron is estimated to be 1000 times lower than for trichlorfon. Out of this, a lifetime cancer risk number is generated.

b. Trichlorfon: Even though the EPA says there are not enough data available to do a quantititive cancer risk assessment of trichlorfon (FEIS F15), the FEIS authors do one. Their results assure the decisionmakers that the risk is about 10 times lower than smoking 2 cigarettes (FEIS p.67). Surely, if the data is too inconclusive for the EPA, this optimistic projection does not qualify as a "worst case" scenario.

c. Acephate. The estimate that the risk of getting cancer from an acephate gypsy moth program is less than living in a brick house for two months (FEIS p.48) is not based upon any scientific, publicly-available study, but rather is based upon Chevron Chemical Company's description (i.e., "personal communication to USDA," FEIS F19) of Chevron's acephate cancer study. It is absurd to base such important conclusions on personal communications from someone with such a vast economic interest, particularly when no data are submitted to support the conclusion.

4. The FEIS cancer risk estimates are based on the assumption that a person will be exposed to either 2 eradication projects (3 exposures/project = 6 exposures) or 10 suppression projects (1 exposure/project = 10 exposures) in a 70 year lifetime. No historical data are used to analyze whether this estimate is accurate, even though such data could have been located by the authors, given the long history of gypsy moth control programs in the United States.

California has been undertaking gypsy moth eradication programs for only eight years. Already, the same location in San Jose, California has received its 70-year dose of chemical insecticides for gypsy moth eradication in two years (i.e., 6 exposures). Los Altos, California, has also run up its 70-year dose in two years (i.e., 6 exposures). (Steve Driestadt, Citizens for a Better Environment, personal communication).

Asked by the California Department of Food and Agriculture to analyze the potential economic impact of the gypsy moth in California, the Giannini Foundation concluded that an effort to suppress the gypsy moth population in Santa Barbara County would involve two spray applications a year for six years in a row out of 20 years (i.e. exceeding within 6 years the FEIS estimate of 10 applications in 70 years for suppression projects). (Galt et al. 1982).

It is clear from both California data and projections that the FEIS "worst case" assumption on exposures is not only false, it is substantially incorrect.

VI. THE ARRANGEMENT OF INFORMATION IN THE FEIS IS DYSFUNCTIONAL

All four insecticides are described simultaneously throughout the text. This renders the FEIS essentially unreadable and useless for meeting the intent of NEPA to allow comparisons of alternatives. Moreover, it makes difficult the identification of data gaps that need to be filled in the interests of reasoned decisionmaking pursuant to 40 CFR 1502.22.

A. A coherent picture of any one insecticide is nonexistent. The cancer risk of acephate, for instance, is summarized on p. 17, discussed on p. 48, and summarized with different information on F15. The cancer potency model is described on F16-F17, the potency calculation for acephate on F19-F20, the cancer risk assessment formula on F73-F74, the assessment calculation for acephate on F80-F81, the expected cancer incidence calculation on F82-F83, and the incidence associated with accidents on F86. The meaning of the estimated risk of acephate-induced cancer is discussed on F95. Meanwhile, all cancer calculations for the other three insecticides are carried out on nearby pages. The cancer discussions and calculations are interspersed with discussions and calculations for all other effects for all the insecticides. The net result is a confusion of important data, obscured beyond the point of utility.

B. No delineation is made for estimates based on hard data and those based on soft data. No-one, for instance, could compare the value of data for each of the insecticide cancer risks (see above paragraph and Part V, paragraph 3). Few readers will notice that the entire calculation of risk of nongenetic effects of diflubenzuron exposure is produced without one piece of experimental or field data on diflubenzuron.

The majority of conclusions in the FEIS do not inform the reader whether they are based on direct, peer-reviewed studies, on unpublished papers by chemical companies or user agencies, or on some type of extrapolation from allegedly related information. At a minimum, the FEIS should directly state what data is publicly available and peer-reviewed. Without that delineation, the FEIS is essentially meaningless in helping decisionmakers draw conclusions about the proposed projects.

A final example perhaps epitomizes this problem. The FEIS states that an "extensive review of human exposure to carbaryl" indicates that "the safety record of carbaryl is almost unparalleled by any other insecticide (FEIS p.53). The "review" is an unpublished Union Carbide paper. Thus, this obviously biased, non-peer-reviewed document is used to suggest an ultimate safety, and is at best misleading in the manner it is presented in this document.

C. The prioritizing of research to resolve uncertainty is made impossible. Because calculations and estimates for any one pesticide are scattered throughout the FEIS, the decisionmaker cannot identify which gaps might be important and yet inexpensively filled or which data gaps must be filled (no matter what the cost) before massive public exposure should take place. It is absurd to make important decisions on the costs of filling data gaps based upon Dow Chemical Company estimates (FEIS40). A skin absorption test would help resolve the uncertainty surrounding human exposure to the three insecticides for which no such data exists (i.e. acephate, trichlorfon, and diflubenzuron). A relatively simple crop vegetation analysis would resolve whether initial residues of carbaryl will be 500 ppm (as documented on maple trees, FEIS p. 49) or 10 ppm (as predicted by the FEIS, p. 43).

If research needs are not prioritized, the cost of filling all data gaps will always appear to be exorbitant, there will be no

incentive to fill any of them, ignorance will be bliss and run rampant, and these types of spray projects will continue without anyone knowing the fate to which they may be condemning future generations.

CONCLUSION

This FEIS systematically underestimates, downplays, and ignores risks associated with gypsy moth insecticides and is unusable either for comparing relative risks of various alternatives or for facilitating orderly acquisition of critically-needed missing information.

Decisionmakers don't need a worst case analysis containing highly technical information that applies and manipulates scientific data inconsistently. Rather, the worst case analysis should first describe documented health effects associated with each pesticide. Next, the probability that these effects will occur should be estimated, when such estimates can be meaningfully made. Finally, the limitations of the data should be acknowledged and, when necessary, the inability to generate meaningful numbers should be admitted.

This FEIS intermixes effects and risks, science and guesses. It so completely ignores scientific accuracy as to be virtually useless. If this obfuscated FEIS is judged adequate to fulfill NEPA requirements, the public and the environment will be ill-served.

Respectfully Submitted,

Mary Hallie O'Brien
Mary Hallie O'Brien

References:

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Letter 17

STATE CLEARINGHOUSE
State of Ohio - Office of Budget and Management

30 EAST BROAD STREET • 39TH FLOOR • COLUMBUS, OHIO 43266-0411 • (614) 466-0697 / 0698

Date: 85-12-09

RCVD. FOSS 12/12/85

US DEPT OF AGRICULTURE, APHIS-PPQ
FEDERAL BUILDING, ROOM 663
HYATTSVILLE MD 20782-0000

Attention: GARY E. MOOREHEAD, S. O. Phone: (301)436-8261

RE: State Clearinghouse Intergovernmental Review-Application Clearance Letter
Project Description: GYPSY MOTH SUPPRESSION & ERADICATION PROJECTS, DRAFT ADDENDUM-FINAL EIS AS SUPPLEMENTED-1985, REFER TO OLD SAI NUMBER 35-445-0011

SAI Number: OH851030-E738-36445
Proposed Federal Funding: $00

The State Clearinghouse (Single Point of Contact) has reviewed the application for the above identified project that is covered by the Intergovernmental Review Process (Presidential Executive Order 12372) and Gubernatorial Executive Order authorized under Ohio Revised Code, Section 107.18(A).

Following the guidelines of Presidential Executive Order 12372 and Ohio's Intergovernmental Review Process, this application has been simultaneously reviewed by the impacted Area Clearinghouse(s) and other interested agencies.

As a result of our review we have determined that your application is consistent with State or local plans, programs, and objectives. Therefore you should proceed with your application to the appropiate funding agency.

A copy of this clearance letter should be attached to your application. In addition, the State Application Identifier (SAI) Number noted on the top of this form must appear as item number 3 on the Federal Standard Notification Form 424, which is a part of your application.

The results of this review are valid for one year. A continuation or renewal application must be submitted to the State Clearinghouse and impacted Area Clearinghouse(s) annually. An application not submitted to the funding agency, or not funded within one year after completion of this review, must be resubmitted to receive a valid intergovernmental review.

Sincerely,

Leonard Roberts Jr

Leonard E. Roberts, Deputy Director
Office of Budget & Management

Letter 18

TENNESSEE VALLEY AUTHORITY
KNOXVILLE, TENNESSEE 37902

DEC 6 1985

RCVD. FOSS 12/12/85

Mr. Gary E. Moorehead, Staff Officer
U.S. Department of Agriculture
Animal and Plant Health Inspection
  Service - PPQ
Federal Building, Room 663
Hyattsville, Maryland 20782

Dear Mr. Moorehead:

This is in reference to the U.S. Department of Agriculture's Draft Addendum to the Final Environmental Impact Statement (FEIS) on Gypsy Moth Suppression and Eradication Projects in the United States as Supplemented 1985.

Based upon our review of the draft addendum, we believe that this plain language version of Appendix F of the FEIS is indeed readable and understandable, and thus meets the requirement for clarity. We have no other substantive comments regarding the information and material presented in this document. With this addendum, we think that the FEIS adequately assesses the consequences of the proposed action.

Thank you for affording us the opportunity to review this addendum. If we can be of any further assistance, please call Charles Langdon of my staff at FTS 856-6693.

Sincerely,

Martin E. Rivers

Martin E. Rivers, Director
Environmental Quality

An Equal Opportunity Employer

Letter 19

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

OFFICE OF
EXTERNAL AFFAIR

DEC 12 1985

12/12/85

Mr. Gary E. Moorehead
Staff Officer
USDA - APHIS - PPQ
Federal Building, Room 663
Hyattsville, Maryland 20782

Dear Mr. Moorehead:

In accordance with our responsibilities under the National Environmental Policy Act (NEPA) and Section 309 of the Clean Air Act, the Environmental Protection Agency (EPA) has reviewed the Draft Addendum to the Final Environmental Impact Statement (EIS) as Supplemented (1985) for the Gypsy Moth Suppression and Eradication Projects. This addendum provides a plain language version of the worst case analysis contained within the Final EIS.

EPA believes that the document accomplishes its purpose of discussing in non-technical language, the "worst case" risk scenarios presented in Appendix F of the Final EIS. The discussion of complex risk assessment issues is readable, clear, and a fair presentation of what we know about the health effects and associate risks of the four pesticides evaluated. EPA does, however, request that changes be made in several locations in the document to more accurately reflect our responsibilities under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA). These are discussed below. Additional comments related to the discussions of specific pesticides are enclosed.

EPA requests that the statement on page H-1, first bullet, be changed to read as follows:

a

> "All realistic doses to the general public from routine spraying would be unlikely to pose any significant risks of adverse effects, according to the evaluations of these chemicals made by the Environmental Protection Agency or the World Health Organization."

b As currently drafted the statement may imply a determination of safety that is more absolute than appropriate. Since pesticides are inherently biologically active compounds, exposure to them always involves some element of potential risk. Thus, any discussion of pesticide use necessarily concerns degrees of risk or probability that significant risks will or will not occur, rather than choices between absolute safety on the one hand or unacceptable risks on the other. We suggest that such a contextual statement be made early in the document, either in the paragraph introducing the conclusions (page H-1), method (p. H-2) or worst case assumptions (p. H-3).

c Correspondingly on page H-10, under the section entitled, "Hazard Levels of the Four Insecticides," we suggest that the second sentence be changed to read as follows:

> "This means that, in EPA's judgment, available studies indicate that none of these chemicals are likely to cause . . . "

Thank you for the opportunity to comment. Should you have any questions concerning our comments please contact Margaret Schneider (382-5070) of my staff.

Sincerely,

Deputy Director
Allan Hirsch
Director
Office of Federal Activities

Enclosure

cc: Fred Honing, U.S. Forest Service

Letter 19
(continued)

Specific Comments on the Draft Addendum
to the Gypsy Moth EIS

Acephate

d An EPA peer review group has recently classified acephate as a possible human oncogen in category C of the proposed Agency guidelines for cancer risk assessment. Category C is "limited evidence." This is a tentative classification, not an official Agency position at this time. Thus, it is not clear that EPA will make any future regulatory decisions based on the assumption that acephate is a carcinogen. However, for purposes of a risk estimating exercise such as the EIS, we would use the latest cancer potency factor (0*) of 9.1 x 10-3 is used by OPP. This is a lower potency than cited in the document (e.g. p. H-12 cites .025 mg/kg/day; our figure would be .009).

e In the discussion of the toxicology of acephate on page H-10, we suggest that the second sentence be changed to read as follows:

> "Its main health effect is to reduce the active levels of cholinesterase, an enzyme found in . . . If this enzyme is bound up by the pesticide (called cholinesterase inhibition) and thus made non-available to the nervous system, a number of ill-health effects can occur."

Cholinesterase inhibition usually means that the enzyme, acetylcholinesteras is bound or tied up by the inhibitor (in this case, acephate), rather than not being produced by the organism. This results in diminished activity of the enzyme at receptor sites, rather than diminished quantities of it in the body. The statement as currently drafted may be read to indicate that the mechanism of action of acephate is to prevent production of cholinesterase, rather than to bind the enzyme.

Trichlorfon

f It is not clear how USDA derived a cancer potency number for this chemical in the absence of any positive evidence of cancer effects, nor is this explained in the "clarification" section of Appendix I. As we understand it, USDA used cancer potency for a surrogate chemical (1,1,1, trichloroethane). If this is the case, it should explain this in Appendix I.

g On page 14 we suggest that more specific mention of the mode of action of the pesticide be made to give the reader more basic comparable information about toxicity. Trichlorfon exerts its primary toxic effect via cholinestease inhibition.

h This could be similarily stated for carbaryl on page 12. The major mode of action here is again cholinesterase inhibition, although a more reversible variety than either acephate or trichlorfon.

Letter 20

UNION CARBIDE

UNION CARBIDE AGRICULTURAL PRODUCTS COMPANY, Inc.
P. O. BOX 12014, T. W. ALEXANDER DRIVE
RESEARCH TRIANGLE PARK, N. C. 27709
(919) 549-2000

December 10, 1985

12/17/85

Mr. Gary E. Moorehead
Staff Officer
USDA-APHIS-PPQ
Federal Building, Room 663
Hyattsville, MD 20782

Dear Gary:

I have completed my review of the Draft Addendum to the Final Environmental Impact Statement on Gypsy Moth Suppression and Eradication Projects in the United States as Supplemented-1985. As discussed in the introduction, this Addendum was written to clarify the worst case analysis presented in the previous Final EIS (Appendix F). I think those of you who participated in this endeavor should be congratulated -- the document is quite readable and provides a better understanding of the risk calculations used to develop a worst case scenario.

Although this draft is well written and easily understood, please note the attached comments with regard to references dealing with SEVIN® brand carbaryl insecticide. Once again, thank you for allowing us the opportunity to review this manuscript.

Sincerely,

Jack Boyne
J.V. Boyne, Ph.D.
Program Coordinator
Foliar Insecticides Group

2484s

INTERNATIONAL TELEX NUMBER 420542—ANSWERBACK UCC UI   REPLYING BY TELEX STATE REFERENCE "RTG"   DOMESTIC—TWX 710 581 5169

Letter 20
(continued)

COMMENTS REGARDING THE DRAFT ADDENDUM TO THE FINAL EIS - 1985

	PAGE	COMMENT
a	H-3	The assumption that a chemical can cause cancer under a worst case scenario when available evidence suggests that it does not is extremely speculative. The tendency to establish cause and effect relationships when none actually exist may contribute to greater misunderstanding from the general public.
b	H-3	The assumption that a person might eat fruit or vegetables tainted with carbaryl residues (following an application to control gypsy moth) is, of course, untrue. Market Basket Surveys conducted in the Unites States by the FDA have demonstrated that dietary exposure to carbaryl in foodstuffs ranged from undetectable to a maximum of 0.088 ug/kg/day (Gartrell et al., J. Assoc. Off. Anal. Chem., 68:842-61, 1985).
c	H-11	The use of N-nitrosocarbaryl to determine cancer potential instead of carbaryl (which, as stated, is considered to be noncarcinogenic) is misleading. Since carbaryl is not considered to be a significant constituent in the human diet, and since it must be ingested to permit its supposed conversion into nitrosocarbaryl, it cannot pose a cancer risk to the general populace. Moreover, the above statement assumes that conversion can occur in the human stomach, an assumption that has not been demonstrated. As Richard and Dorough (J. Tox. Env. Health, 14:279-90, 1984) states, "It might be assumed, therefore, that if nitrosocarbamates are indeed formed in the stomach of the general population, the incidence of stomach cancer would show an increase over the past 20 years as a result of heavy carbamate insecticide use. However, the incidence of gastric cancer in the United States has declined considerably during this period."
d	H-35	The assumption that carbaryl might pose some risk of birth defects to sensitive individuals under worst case conditions is not valid, considering that dose exposures would be 1,000 times below the lowest non-dog NOEL for birth defects and that carbaryl is not considered to be a human teratogen.
e	H-37	Table H-9 estimates the weighted risk of cancer to an individual if exposed to different insecticides over a lifetime. As you stated, for carbaryl, the only doses relevant to cancer are those obtained through eating and drinking. Therefore, I assume the 1,000 value listed under "Truck Spill - dermal" is a typographical error.

Letter 21

[illegible] 12/17/85

COMMONWEALTH *of* VIRGINIA

Council on the Environment

KEITH J. BUTTLEMAN
ADMINISTRATOR

903 NINTH STREET OFFICE BUILDING
RICHMOND 23219
804-786-4500

December 12, 1985

Mr. Gary E. Moorehead
Staff Officer
U.S.D.A - A.P.H.I.S. - P.P.Q.
Federal Building, Room 663
Hyattsville, Maryland 20782

Dear Mr. Moorehead:

The Commonwealth of Virginia has completed its review of the Draft Addendum to the Final Environmental Impact Statement as Supplemented on the Gypsy Moth Suppression and Eradication Projects. The Council on the Environment is responsible for coordinating Virginia's review of federal environmental documents and responding to appropriate federal officials on behalf of the Commonwealth. The following agencies took part in this review:

a Department of Agriculture and Consumer Services
Department of Conservation and Historic Resources
Department of Health
Virginia Water Control Board.

The plain-language summary of the earlier health risk analysis and clarification of toxicity information for the four chemicals dicussed in the earlier document have aided our understanding of the issues associated with chemical controls used in gypsy moth suppression and eradication. Thank you for the opportunity to review this document.

Sincerely,

Keith J. Buttleman

Keith J. Buttleman

Attachment

cc: The Honorable Betty J. Diener
Mr. Donald H. Kludy, DACS
Mr. C. E. Easlick, VWCB
Ms. Bonnie S. Greenwood, DCHR

Letter 21
(continued)

COMMONWEALTH of VIRGINIA

S. MASON CARBAUGH
COMMISSIONER

BILLY W. SOUTHAL
DIRECTOR

DEPARTMENT OF AGRICULTURE AND CONSUMER SERVICES
Division of Product and Industry Regulation
P. O. Box 1163, Richmond, Virginia 23209

NOV 1985
Council on the Environment

November 15, 1985

Mr. Charles Ellis
Council on the Environment
903 Ninth Street Office Building
Richmond, Virginia 23219

Dear Mr. Ellis:

I have reviewed the Draft Addendum to the Final Environmental Impact Statement as Supplemented - 1985 for Gypsy Moth Suppression and Eradication Projects. This addendum includes Appendix H: Plain Language Summary of the Health Risk Analysis and Appendix I: Clarification of Information About the Toxicity of Acephate, Carbaryl, Diflubenzuron and Trichlorfon. The addendum was written to meet the regulatory requirement for clarity.

b

The document is much clearer and easier to understand than the Worst Case Analysis (Appendix F) found in the Final Environmental Impact Statement as Supplemented - 1985 for Gypsy Moth Suppression and Eradication Projects dated March 8, 1985. The assumptions necessary to determine a worst case analysis are reasonable, a necessary prerequisite for the rest of the analysis.

Sincerely,

Donald H Kludy

Donald H. Kludy
State Entomologist & Chief
Bureau of Plant Protection
and Pesticide Regulation
804/786-3515

DHK/cbf

Letter 22

STATE of INDIANA

INDIANAPOLIS, 46204

DEPARTMENT OF NATURAL RESOURCES

JAMES M. RIDENOUR
DIRECTOR

December 16, 1985
Vallonia State Nursery
Vallonia, IN 47281

Gary E. Morehead, Staff Officer
USDA - APHIS - PPQ
Federal Building, Room 663
Hyattsville, Md 20782

Dear Gary:

I received the draft addendum to the FEIS on Gypsy Moth Suppression and Eradication Projects, Appendix H and I on December 11, 1985. Our in-house mail created this delay. Although I am late for the responce deadline. I submit the following comments for your consideration.

a My initial thought after reading the plain language version of appendix F (appendix H) was 'What does the plain language version tell me?' Is it suppose to make definte statements on health risk from the four chemicals? If it is to do this, they should be extracted and made easier to identify from the general text.

b I felt disorganized in following the outline of the appendix H. Headings were made in the left hand margin or above a paragraph or section. I could not tell the importance of the headings. I suggest that the four basic sections - Overview, Hazard Identification, Exposure Analysis and Risk Evaluation be reformated or laidout in the text of appendix H to know that they start 'here' and stop 'there'. Again, with subsectins within the above four sections, they also need to be better identified as to when they start and stop. I found myself referencing back to see what section or subsection the text pertained to. Trying to differeniate sections and subsections by using all caps for section titles, underlined subsections and single double spaced lines for sub-subsections within the text margins was confusing. Also the single double spaced lines for sub-subsections (i.e. page H-15 'Exposures from routine spraying operations') appeared to be part of the text.

c Page H-27, the last paragraph on carbaryl, for the last sentence of the paragraph, it explains how to get the average lifetime dose. This needs to have reference to table H-7 added to the end of the sentence. If appendix F, shows an example of how to calculate this, do not include an exampole but include and exampole if it doesn't.

"EQUAL OPPORTUNITY EMPLOYER"

Letter 22
(continued)

d Page H-28, the last sentence of the top paragraph 'The total doses are than calculated just as for carbaryl'. Change this sentence to actually describe the calculation as was done for carbaryl on page H-27. Also for diflubenzuron change the last sentence of the section to explain the calculations as for carbaryl. Examples of the calculations for both would help.

e For table H-8. A clearer explanation or ference is needed to explain that this table is derived from comparisons of tables H-1 to H-4 and H-5. This reference should be included in the text on page H-30 or as footnote to table.

f On page H-33 when the statements say '6 times above the ADI and half the lowest NOEL', reference these numbers to tables H-1 to H-4 and H-5 (give example of calculation). Continue this reference to table H-1 to H-4 and H-5 on pages H-34, H-35, and H-36 where needed in text. This will help to explain where the number '6 times' was obtained.

g Where is the table listing the birth defect NOEL for each chemical? In appendix F? If it is not in appendix F, it needs to be in appendix H.

Respectfully submitted,

Philip T. Marshall
Foerst Pest Specialist

Letter 23

NCAP
NORTHWEST COALITION for
ALTERNATIVES to PESTICIDES
P.O. BOX 1393 EUGENE, OREGON 97440 (503) 344-5044

December 18, 1985

12/23/85

Gary E. Moorehead, Staff Officer
USDA - APHIS - PPQ
Federal Building, Room 663
Hyattsville, MD 20782

Dear Gary Moorehead,

This letter is regarding the Northwest Coalition for Alternatives to Pesticides' Comments on the Draft Addendum to the Final Environmental Impact Statement on Gypsy Moth Suppression and Eradication Projects in the United States as Supplemented - 1985. We request the following change be made in your copy of the document:

page 18, Comment 43 (C43)

current sentence reads--
"Estimated doses that are the same as or below the ADI are predicted to pose no carcinogenic or mutagenic heath risks."

intended sentence should read--
"Estimated doses that are the same as or below the ADI are predicted to pose no nongenetic health risks. The ADI does not indicate whether risk of cancer or genetic damage is present."

Thank you for your assistance in this correction.

Sincerely,

Mary O'Brien

Mary O'Brien
Information Coordinator
NCAP

RESPONSES

Specific responses to comment letters follow. Unless otherwise indicated, references to pages in Appendixes H and I refer to page numbers in this Final Addendum. Several of the responses refer to District Court Judge Redden's Opinion in Oregon Environmental Council v. Kunzman (OEC v. Kunzman), as well as to the Narrative Witness Statement of John Neisess before the Court. Judge Redden's Opinion (Federal Supplement, Volume 614, Number 2, pages 657-666) and the statement of Dr. Neisess are included in this appendix to aid the reader; they begin on pages J-138 and J-143, respectively.

Letter 1 — No response required.

Letter 2 — No response required.

Letter 3 — No response required.

Letter 4 — No response required.

Letter 5 — No response required.

Letter 6 — No response required.

Letter 7 — No response required.

Letter 8 — No response required.

Letter 9 — No response required (attachment not received).

Letter 10 — No response required.

Letter 11 — No response required.

Letter 12 — No response required.

Letter 13 — No response required.

Letter 14 — No response required.

Letter 15

a Figure H-4 shows an observer using a pair of binoculars to observe spraying operations, not drinking water.

b We agree that there were some places in the Draft Addendum where readability could be further improved. We have carefully scrutinized the text and made many improvements. We used your specific suggestions for improving readability where appropriate; for example, we added more cross-references to Appendix F to aid readers in finding more detailed discussions. Please keep in mind that we were constantly striving to balance the need for accurate information with the need for clear prose. Some commenters (such as you) tugged us in the direction of simplifying the discussion. Others (for example, letter 22, comment d) tugged us toward using more technical language or mathematical computations. We believe that reasonable people will agree that we have provided a faithful and readable summary of the health risk analysis.

Letter 16

a The Final Environmental Impact Statement (FEIS) as supplemented with the Final Addendum is one legal document. The Addendum was written in response to OEC v. Kunzman, which held that Appendix F of the FEIS did not meet the readability requirements of the National Environmental Policy Act (NEPA) regulations (40 CFR 1502.8). (See pages J-138 to J-142 for the full text of Judge Redden's Opinion.) The purpose of the Addendum was to provide the decision-maker and the public with a plain language version of the worst case analysis (Appendix F) in the FEIS. This Final Addendum should be read with Appendix F. Those who wish to examine a more technical documentation supporting the worst case analysis should concentrate on Appendix F.

b In his Opinion, District Court Judge Redden states that "the text of the FEIS (pages i-78), although technical, does meet the requirements of § 1502.8" (OEC v. Kunzman, page 665). Therefore, USDA focused corrective measures on the readability of the worst case analysis contained in Appendix F of the FEIS.

c The argument about doing research to fill data gaps was raised before the District Court in OEC v. Kunzman. Data gaps and the cost of conducting research to fill the gaps were analyzed on pages 38 to 42 of the FEIS. Other data gaps were identified in Appendix F (see, for example, F-27 where available exposure data are discussed). The District Court found that data gaps and the need for research were properly addressed in the FEIS. The court concluded that "it was reasonable for the agency to prepare a worst case analysis" (OEC v. Kunzman, page 663).

d The FEIS discussed the estimated incidences of cancer (number of people getting cancer) on pages F-95 and F-96 of Appendix F. Because future activities are uncertain, these estimates were based on the historical data regarding the number of acres treated with each insecticide. In holding that this subject was adequately covered in the FEIS, the District Court in OEC v. Kunzman held that to require an agency to "set an overall specific estimated number of cancer victims is unreasonable" (OEC v. Kunzman, page 664). Appendix F also provides equations for calculating risk once specific data on acres treated are known (page F-82). Possible incidences of cancer are summarized on pages H-38 through H-41 of the plain language version of the risk analysis.

Risks associated with multiple applications of spray in a single season are addressed in the FEIS and Final Addendum. In preparing the risk data for lifetime exposure, multiple spray applications during a lifetime are considered. These figures then are used to calculate lifetime risk. Additionally, a member of the public or a decision-maker may always assume the risk in a single year to be substantially less than the risk over a lifetime of exposure.

e USDA has adopted the most recent guidelines for analyzing chronic health risks. The District Court ruled on the use of NOELs, ADIs, and safety factors. The District Court opinion states that "there was testimony that the use of these figures tends to overestimate associated risks to humans. Thus, the use of the figures to estimate human health risks is reasonable and does not render the document inadequate" (OEC v. Kunzman, page 663). Because it is important to understand the use of NOELs and ADIs when evaluating possible health effects in humans, these concepts have been discussed further in the plain language version of the risk analysis (pages H-5 through H-8).

f This same argument about sensitive individuals also was presented before the District Court. The FEIS states on page F-99 that NOELs were reduced by a factor of 100 for evaluating health risks to sensitive individuals. For example, the NOEL of 3.125 mg/kg/day for carbaryl that was used for evaluating risks to the general public was reduced to 0.03 mg/kg/day for the sensitive population. The FEIS (page F-100) concluded that the margins of safety for sensitive individuals exposed to trichlorfon, acephate, and carbaryl were less than 100. Therefore, adverse health effects could result from exposure to these insecticides. The Court ruled that "the FEIS adequately discussed the potential risks to chemically sensitive persons" (OEC v. Kunzman, page 662). Risks to sensitive individuals also are discussed in the Risk Evaluation section of Appendix H, on pages H-30 through H-38.

g The health risk analysis (Appendix F) does not conclude that any exposures are "safe" or "unsafe." Its purpose is to provide data that enable the decision-maker to weigh risks and benefits in order to reach a conclusion about what means, if any, should be used to suppress and eradicate gypsy moths.

C1 This statement is made in the context of ADIs, which are set by the Environmental Protection Agency (EPA) and the World Health Organization (WHO), and it relates to threshold effects only. Cancer is a nonthreshold effect and no such statement regarding nonthreshold effects is made in this document. The sentence on page H-1 has been modified in response to comment 19a from EPA. The sentence now indicates that realistic doses are unlikely to pose significant risks to humans.

C2 The statement that you refer to has been modified. In response to your concern about "underestimations of exposure" and "unscientific extrapolation procedures," we offer an excerpt from the testimony of Dr. Edward J. Calabrese, Professor of Toxicology at the University of Massachusetts School of Public Health, presented during OEC v. Kunzman: "The approaches used in extrapolating acceptable levels of exposure in the FEIS were designed to be consistent with the approaches developed by the most highly regarded organizations in the United States concerned with environmental risk assessments" (Narrative Witness Statement of Dr. Edward J. Calabrese, page 4). Smoking as a cancer benchmark was chosen because of the public's ability to relate to this practice and its association with cancer.

C3 This conclusion is substantiated by a review of the published literature presented on pages F-12 to F-14 and summarized in the Hazard Identification section of Appendix H (pages H-11 to H-15). The weight of evidence showed that mutagenic responses that were found in bacteria or single mammalian cells were not generally found in whole animals. Because trichlorfon showed a potential to reach reproductive cells in humans, a worst case probability of heritable mutations was estimated (pages F-97 and F-98). The probability was 0.1 chance in a million for possible heritable mutations resulting from exposure to trichlorfon. We consider this to be an unlikely event.

C4 We believe that the statement "this analysis assumes the worst" is accurate. In the example mentioned (regarding the cancer potency of N-nitrosocarbaryl), the higher cancer potency figures are presented on page H-14. A more detailed response to this comment is presented in the response to comment C19.

C5 The sentence is not misleading; it is intended to provide some perspective on the use of worst case assumptions. The sentence would be misleading if it led readers to think that the four chemicals cannot cause cancer. But the preceding sentence clearly indicates that we assumed that the chemicals could cause cancer. Adding the sentence that you suggest would reduce the readability level by inserting a double negative, and it would introduce an unverifiable assertion (it is impossible to prove that the four chemicals have not caused cancer).

C6 The statement on page H-5 of the Draft Addendum (now on page H-6) has been changed to indicate that we tried to use the lowest NOEL in the health risk analysis. The caption on Figure H-2 also has been changed to reflect that the lowest NOEL is not always used to set an ADI. With one exception, the lowest NOEL was used in the risk analysis; the exception was carbaryl. The lowest NOEL for carbaryl used in Appendix F was 3.125 mg/kg/day. During the hearing before the District Court (OEC v. Kunzman), the plaintiffs pointed out that the NOEL of 0.06 mg/kg/day had been omitted. Because USDA had inadvertently omitted all carbaryl NOELs for cholinesterase inhibition or kidney dysfunction from Table 2, page F-115, this information was added to Appendix I. The omission of the data was discussed in the Court hearing (see Narrative Witness Statement of John Neisess, page 4). The omitted toxicity data were added to Appendix I so that decision-makers and the public were given the opportunity to review data that were clarified for the Court.

USDA did not use the NOEL of 0.06 mg/kg/day to set an ADI for carbaryl because USDA did not set the ADI. ADIs used in the risk analysis came from the Environmental Protection Agency, which has the responsibility for setting such threshold values in the United States, as well as from the World Health Organization. In the Addendum, we chose to continue to use the NOEL of 3.125 mg/kg/day (birth defects in dogs) as the low NOEL because this was a more conservative figure than the NOEL of 10 used by EPA to set the ADI. If studies now being conducted by the registrant resolve the issue of the sensitivity of dogs compared to other mammals, or if EPA were to set lower NOELs or ADIs, these new values would be incorporated into future revisions of the FEIS.

Regarding your final comment about the use of NOELs and LOAELs, see the response to your comment C7.

C7 Page F-87 of Appendix F clearly describes the manner in which the ADI is used to help identify potential health risks. Nowhere in that document (nor in Appendix H) are doses that exceed the ADI ignored. Rather, Appendix F (page F-87) states that "doses calculated as equal to or below the established ADI are considered to be within a margin of safety by regulatory agencies in the United States and worldwide for the general population. . . . For doses that exceed the ADI, it is necessary to examine the toxicological data base more closely."

USDA provided extensive testimony in the District Court (OEC v. Kunzman) regarding the use of ADIs and NOELs in assessing human health risks. There also was testimony that the use of these figures tends to overestimate associated risks to humans (see response to comment C2). The District Court, regarding the use of ADIs and NOELs, ruled that "the use of the figures to estimate human health risks is reasonable and does not render the document inadequate" (OEC v. Kunzman, page 663).

The ADIs used in the health risk analysis were established by EPA. If future toxicological information becomes available that leads EPA to establish a new ADI, the risk analysis will be changed to reflect that new ADI.

C8 Figure H-3 is provided only as a general visual aid. However, the curved line provides a reasonable fit for the acephate data presented on pages F-19 and F-20. During the trial OEC v. Kunzman, Dr. Edward J. Calabrese, Professor of Toxicology at the University of Massachusetts School of Public Health, testified that "the models selected by the Federal Government for low-dose cancer risks were chosen in part because of their highly protective or conservative nature. Thus, the Government's attempts to predict cancer risks at low doses are designed to err on the side of safety. An example of the protective nature of the Government's practice was put forth by Carlborg (1979). He reported that the EPA has estimated via the use of the one-hit model that current human exposure to DDT, dieldrin, and aflatoxin are responsible for 153,000 liver cancers per year in the United States. However, there are about 7,000 to 8,000 actual liver cancers per year in the entire United States from all sources of exposure" (Narrative Witness Statement of Edward J. Calabrese, page 9). Statements such as this one prompted USDA to provide a visual aid that indicates how linear extrapolation may overestimate risks at low doses in order to make this concept more understandable for the decision-maker and the public.

C9 The 50-percent cancer probability figures were included to help readers comprehend cancer potencies. We felt that one chance in two was easier to grasp than one chance in a million. But, other than making the statistics readily understandable, the 50-percent figures probably served no practical purpose and confused the discussion. We therefore have deleted them.

C10 We disagree with your criticism of the use of the linear cancer model to predict mutations. The linear cancer model was used to overstate the risk of mutagenesis because there are no generally accepted models available to estimate the risk of mutagenesis. According to personal communications from Dr. David Brusick, head of all toxicology testing programs at Litton Bionetics, Incorporated, it is safe to assume that a linear cancer model will overestimate the risk of mutagenicity. This is because there are more somatic cells than germ cells. The rationale for the assumption is explained on pages F-97 and F-98 of the FEIS.

C11 The sentence in question on page H-10 of the Draft Addendum has been changed in response to a comment from EPA (Letter 19, comment c). The sentence, which now appears on page H-11, has been changed to read as follows: "This means that, in EPA's judgment, available studies indicate that none of these chemicals are likely to cause unreasonable adverse effects in people or the environment when properly used." Also see comment 19a and our response.

C12 The sentence you refer to has been changed to read as follows: "The acceptable daily intakes, no-observed-effect levels, and acute lethal doses used in Appendix F are compared in Table H-1." The caption for Table H-1 also has been changed accordingly. See the response to comment C6 for an explanation of why 3.125 mg/kg/day was used as the lowest NOEL.

C13 The sentence you refer to regarding acephate's mode of action has been changed in response to a comment from EPA (Letter 19, comment e). In addition, a discussion of the metabolite methamidophos has been added to Appendix I (page I-1). The cited study (Savage, Eldon P.; Lewis, James A.; Parks, Leland H. Chronic Neurological Sequelae of Acute Organophosphate Pesticide Poisoning: An Epidemiologic Study. Final Report. April 1982.) does not involve "large, one-time doses to humans of organophosphate" as the comment states. The study involved 100 individuals who had experienced organophosphate pesticide (OP) poisoning. Seventy-nine percent of the cases involved poisoning by either parathion or methyl parathion. Of the 100 cases, there were 11 where more than one noteworthy organophosphate pesticide poisoning was reported: 8 experienced two poisonings, 1 experienced three poisonings, and 2 reported four poisonings. No quantitative information was available on the exposures to these pesticides. However, the authors pointed out that the study participants received exposures large enough to cause severe illness. Exposure was of such a magnitude as to require hospitalization in 78 percent of the cases. The conclusions from the EPA summary of this study (Summary of an OPP Sponsored Research Project: Organophosphate Poisoning Chronic Neurological and Neuropsychological Effects, April 5, 1985) are printed in full in response to your comment regarding the appearance of "permanent intellectual impairment":

> It is not possible with a single study to state whether or not organophosphate poisoning leads to adverse effects on neurological or psychological function. Several potential sources of bias in the study design may have had important effects on the results. Demographic factors, not neurological or psychological factors, were used to match the case cohort to controls. While the two groups were paired on a demographic basis, they may have differed significantly on a psychological basis unrelated to pesticide exposure. Individuals who work as pesticide applicator[s], mixer

loaders and formulators may differ on certain psychological variables from individuals of similar demographic background who work in other areas.

Statistically significant differences were detected only on the psychological tests. In these tests, an individual's performance or response may have been influenced by the individual's knowledge that he had been poisoned and was being tested for effects. Anxiety factor, rather than organophosphate poisoning, could produce the study outcome.

Since the case and control groups differed significantly in IQ, this could explain other differences found in psychological testing. The case and control cohorts could have been matched, for example, on the basis of childhood IQ scores. From the study data, there is no way to determine if an IQ difference existed before as well as after the poisoning.

It should be emphasized that the effects observed were subtle. None of the poisoned individuals had sought medical care for chronic effects or claimed any noticeable decrease in their intellectual or psychological function as a result of their poisoning.

It is not possible to generalize from the results obtained in this study to the population of individuals who use organophosphate pesticides on a long-term basis but have not experienced a poisoning.

Work is currently underway in the EPA Office of Health Research, Office of Research and Development, to develop more sophisticated methods of detecting neurological and psychological impairment. These methods may be applied in further studies on the chronic effects of pesticides.

If information on neurological or psychological impairment related to acephate or trichlorfon becomes available, the risk analysis will be changed to reflect that new data.

C14 These two sentences were meant to convey the information given in the last two sentences of the complete paragraph on page F-13 in Appendix F. But they inadvertently misrepresented the original statement. We have corrected the sentence on page H-11 of the Final Addendum.

C15 A discussion about ADIs is given on page H-7. USDA used ADIs that were set by EPA or the World Health Organization. If there was a difference between the ADIs set by the two organizations, we used the one from EPA because it is the agency with regulatory responsibility for the United States.

Documentation regarding the ADI for carbaryl is found on page F-67, where the FEIS explains that the ADI is based on a 2-year study using rats. As explained in response to comment C6, this study was inadvertently omitted from Table 2, F-116. That was one of the reasons the information was summarized in Appendix I. The study is cited in Table I-1 (Carpenter et al., 1961). It also is discussed on page I-8 where the 200 ppm dose has been expressed as 10 mg/kg/day.

C16 As you point out, the Halpin (1980) study was not a study of actual exposure to carbaryl. The study examined the rates of birth defects for a 3-year period in municipalities that had been sprayed with carbaryl for gypsy moth and in those that were not sprayed. Although it is not an exhaustive study, it does provide a basis for concluding that there was no association between spraying with carbaryl and birth defects in the sprayed municipalities. The section you refer to in Appendix H has been changed to reflect this lack of association (see page H-13).

C17 As explained in the responses to comments C6 and C12, USDA used ADIs set by EPA or WHO. As pointed out in comment C15 "ADIs are established on the basis of long-term feeding studies." Studies dealing with birth defects generally involve exposure during the gestation period (short term).

The FEIS and Addendum use the NOEL of 3.125 mg/kg/day for birth defects, which is the lowest birth defect NOEL for carbaryl (pages H-13, H-36 and H-37; F-71, F-93, and F-94; Table 7, page F-122; and Table 9, page F-124).

C18 Those studies refer to the two studies using dogs (Smalley et al., 1978; and USEPA, 1980a). There have been a total of 24 teratogenicity studies done with carbaryl. Studies with other species of animals either found no evidence of teratogenicity or produced birth defects only at dose levels high enough to cause toxicity in the mothers. Because the next lowest birth defect NOEL is 150 mg/kg/day for rabbits (Table I-1, pages I-4 and I-5; and Table 2, page F-116), we conclude that "dogs might be much more sensitive than other mammals to this chemical" (page H-13 of the Final Addendum).

C19 Information about the cancer potency of N-nitrosocarbaryl discussed during OEC v. Kunzman is presented in Appendix I. The higher cancer potencies are not ignored; rather, they are presented in the plain language version on page H-14, where they are compared to the lower cancer potency. Risk values that use the higher cancer potencies discussed in Court are calculated in Appendix I on page I-18. These higher risks also are discussed in the plain language version on page H-40.

USDA concluded that the recalculated cancer potencies did not represent significant new information because the cancer risk from N-nitrosocarbaryl, as a result of carbaryl exposure, was determined to be less than 1 chance in a million (see pages H-40, I-18, and I-25). The Record of Decision for the FEIS stated that USDA felt that cancer risks less than one in a million would be acceptable for gypsy moth eradication and suppression programs. Therefore, the recalculated cancer potencies are within the range of risk considered acceptable by the decision-maker in the Record of Decision for the FEIS.

The decision whether or not to prepare a supplemental EIS is within the discretion of USDA. If the agency determines that the circumstances or information are not new, or are not significant, or that the changes are not substantial, the agency need not prepare a supplemental EIS. The modifications that have been made as a result of public comment and internal agency review do not encompass significant new information, nor do they require a substantial change in the proposed alternative or the FEIS. Since there is no substantial change or significant new information presented, the goals of informed public participation and full disclosure have been met. Therefore, USDA declines to prepare a new or supplemental EIS.

C20 See the response to comment C9.

C21 See the correction on page H-15 of the Final Addendum indicating that cancer potencies are calculated in Appendix I. The cancer potency of 4-chloroaniline was estimated in Appendix F to be below that of acephate (see page F-83 in Appendix F, which indicates why this cancer potency was estimated). See the letter from EPA (Letter 19, comment d), which indicates the use of a much lower figure for acephate (0.009, as opposed to 0.025). This once again shows that the risk figures used in the risk analysis tend to overestimate risk to humans.

C22 See response to comment C9.

C23 We disagree. According to personal communication with Dr. Richard Thomas, consulting toxicologist and President of Thomas and Thomas Technologies, Inc., the most commonly reported acute LD_{50} values for trichlorfon range from 400 mg/kg to 650 mg/kg. A number of classification systems are used to rate the toxicity of various chemical substances. The classification system of Maxwell, 1982 (as cited in Walstad and Dost, 1984), classifies pesticides with an LD_{50} of 50 to 500 mg/kg as "moderately toxic."

C24 As indicated in Table H-1 (on page H-12) and in the discussion in Appendix I (page I-14), the lowest NOEL for trichlorfon is indeed 1.0 milligram per kilogram per day. The 1.25 mg/kg/day on page H-14 of the Draft Addendum resulted from an editorial error. The lowest NOEL of 1.0 mg/kg/day was used in the analysis. This error has been corrected.

C25 Appendix I has been changed to clarify the fact that the rats were exposed to a dosing regime of 8 mg/kg/day or a single dose of 80 mg/kg/day (page I-14).

C26 The text of Appendix I (page I-14) has been changed to include the hamster NOEL of 200 mg/kg/day.

C27 The field study in question, "Measurement of exposure to the carbamate carbaryl, Maine carbaryl study, 1978" (SCESC, 1978), was conducted for EPA. The reports of the study can be obtained from that agency. The findings by Keil and Loadholt referred to in the comment were not used by the authors of the SCESC study. The SCESC report states as follows:

> An objective of this study was to estimate exposure (carbaryl dosage) of each participant in the study by utilizing the regression model estimated by Keil and Loadholt (Appendix F). Such a dosage is obtained by substituting the alpha-naphthol value standardized for urine volume per hour in the aforementioned model as follows:
>
> $$\text{Estimated Dosage} = \frac{\text{Estimated alpha-naphthol} - .151}{-.249 + (.0025^2)(\text{weight})}$$
>
> Because all of the alpha-naphthol values, when standardized, were less than .151 and the denominator of the above equation was positive in every case, the estimated dosages were universally negative (usually zero). Hence, it is evident that the level of carbaryl exposure of the participants in this study was far less than the exposure found in the dosing study for which the model used was obtained (SCESC, 1978, page 47).

In other words, SCESC was not able to use the model to estimate carbaryl doses.

The SCESC report goes on to say that another method was used to estimate carbaryl doses: "It was possible to extrapolate estimated dosages by cohort (Table 11). The total alphanaphthol excreted, expressed in µg, was found by multiplying the mean alpha-naphthol (ppm) and the mean volume of urine (cc) for each cohort. This value was then multiplied by a factor of three because the Union Carbide dosing data found that 32.8% (± 6.99) or approximately only one-third of the cumulative carbaryl dose is excreted as alphanaphthol during the first twelve hours." Because we were unsure whether or not the 3 factor allowed for the difference in molecular weights between alpha-naphthol and carbaryl, we increased the dose values by 39 percent. The use of these multipliers was explained on page F-28. We also explained why the dermal absorption rate of 73.9 percent reported by Feldman and Maibach (1974) was not used. This explanation is presented on pages F-29 and F-30. Also see the response to comment C28.

C28 The statement you refer to has been modified to indicate that the absorption rates may be lower (see page H-17 of the Final Addendum). This change more accurately expresses the uncertainty surrounding absorption rates. The rationale for using the 10-percent value was presented on page F-29 of the FEIS. The method for estimating dermal absorption was published in Eto (1977).

There were two reasons why the 73.9 absorption rate was not used in the FEIS. First, Feldman and Maibach dissolved the carbaryl in acetone before applying it to the skin of the test individuals. In USDA projects, carbaryl is suspended in either water or oil. We do not know what effect acetone may have on absorption rates. Second, the use of the 73.9-percent value to estimate exposure results in doses that conflict with other published studies that measured exposure to application groups.

C29 In response to the comment, the paragraph referred to (page H-21) has been rewritten to indicate that, although some fruits and vegetables are growing during the spring when gypsy moth spraying is conducted, none are mature enough at that time to be harvested. In addition, insecticide residues in vegetables degrade rapidly within 1 or 2 weeks. Thus, the likelihood that people would get doses from eating vegetables or fruit with insecticide residues is very low. Nevertheless, to ensure that even this remote possibility is considered, doses from this source of food are included in the dietary dose. Realistic and worst case doses from residues in vegetables and fruits were calculated using studies on residue levels from agricultural applications.

Regarding the comment about fruit and vegetables growing in California, the analysis assumed a dietary component of 0.5 kg of vegetables eaten each day. These vegetables were assumed to contain spray residues. The total dose would not vary much if the diet consisted of both fruits and vegetables because the diet still would consist of 0.5 kg of vegetables and fruits. Fruits would have lower residues because they have greater mass for the same amount of surface area compared to leafy vegetables. The State of California currently does not participate in the gypsy moth eradication and suppression projects covered by this EIS. If cooperative eradication suppression projects are undertaken in the future, the site-specific environmental assessments should discuss the differences in diet between California and other States.

C30 The procedures followed and the assumptions made to determine exposure levels are presented in detail in Appendix F (pages F-26 through F-65) and again in Appendix H (pages H-15 through H-29). Testimony also was provided in District Court during OEC v. Kunzman describing the exposure analysis process and the use of particular data (see Narrative Witness Statement of John Neisess, pages 5 to 8). We have attempted to portray risks accurately by using risk-maximizing techniques so that the "bottom line" risk would not be underestimated. In other testimony before the same court, Dr. Edward J. Calabrese, Professor of Toxicology at the University of Massachusetts School of Public Health, stated that "I have never seen a greater effort made to identify all possible avenues of exposure and all reasonably possible exposure contingencies" (emphasis added). He concluded by saying that the risk analysis "has synthesized the available information in such a way as to consistently try to err on the side of safety" (see Narrative Witness Statement of Dr. Edward J. Calabrese, pages 4 and 12).

C31 The persistence data used to determine lifetime dose estimates are actual residue levels in vegetables and meat from agricultural applications. Residues in water came from studies where the insecticides were applied to control the gypsy moth. This information is presented in Appendix F (pages F-37 through F-43) and Appendix H (Table H-4 and pages H-27 and H-28). Why particular data points were used, and why others were not used, was further clarified in testimony presented in the District Court during OEC v. Kunzman (see Narrative Witness Statement of John Neisess, pages 6-8). We believe that the persistence values used in the analysis accurately portray conditions found during gypsy moth applications. Further, regarding absorption and persistence rates used in the FEIS, the District Court found that USDA's choice of data upon which to rely was within the agency's discretion and was reasonable--not capricious or arbitrary (see OEC v. Kunzman, page 662).

C32 We agree that the number of lifetime exposures to chemical insecticides used in eradication and/or suppression projects are estimates. However, as USDA indicated in District Court (OEC v. Kunzman), these estimates are based upon historical information gathered from sources such as past environmental impact statements and site-specific environmental assessments, State project proposals, project reports, and conversations with State cooperators (see Narrative Witness Statement of Noel F. Schneeberger, page 4). A history of gypsy moth eradication projects, including locations, acres treated, pesticides used, and dates of projects, is presented in Appendix E of the FEIS.

C33 The National Research Council paper that Dr. Ruth Shearer mentioned in OEC v. Kunzman discusses nitrogen oxides as common environmental pollutants produced by combustion. However, the paper does not mention the nitrosation of carbaryl with nitrogen oxides.

Our review of the scientific literature failed to find any studies where nitrogen oxides were reacted with carbaryl in an attempt to produce N-nitrosocarbaryl. The literature does contain studies where carbaryl is nitrosated to form N-nitrosocarbaryl under conditions similar to those in the human stomach (for example, mild acid, 37 °C, and in the presence of nitrite). Therefore, the only dose of carbaryl that was considered was the dietary dose. Another commenter expressed a different point of view regarding the formation of N-nitrosocarbaryl; see Letter 20, comment c.

C34 The data referred to on page 49 of the FEIS represent residue data measured on oak and maple foliage and range grasses. The residue data presented on page H-27 of the Addendum represent residues measured in meat, water, and agricultural crops that would more likely make up an individual's diet. USDA believed that, when evaluating cancer risk, it was not appropriate to extrapolate residue data from forest foliage and range grasses to represent residues that could be present in the human diet when actual residue data were available for the types of food that people normally consume. These residue data are presented and discussed in Appendix F (pages F-37 through F-43) and also were presented and discussed in the District Court during OEC v. Kunzman (see Narrative Witness Statement of John Neisess, pages 6 to 8). Also see responses to comments C30 and C31.

C35 The second sentence in the paragraph you refer to has been changed to read "The first includes those living in the treatment area during spraying and eating food and drinking water having residues (the direct plus dietary exposure scenario)" (page H-28). This describes the group we categorized as "direct," as explained in the FEIS on page F-30. Exposures included secondary exposures "from insecticides residues on grass, foliage, cars, yard items, etc.," as well as possible direct exposure if the resident were outside during spraying. The studies on which the "direct" exposure values were based sampled individuals who were both inside and outside during spraying. Others included in the sample were away from their home during treatment. These individuals would then be exposed only to secondary exposure. The second exposed group considered in the analysis includes people who may be standing directly under the spray aircraft (observer group), who would receive a direct application. Exposure data on this group were based on field studies for the observer group. However, the worst case exposure was calculated based on a direct application landing on exposed skin (see pages F-28 through F-31 in Appendix F).

For an explanation of why USDA did not use the 73.9-percent dermal absorption rate, see the responses to C27 and C28.

C36 The information in Appendix F (page F-43) and Appendix H (page H-28) represented actual residue data from agricultural vegetables treated with acephate. The residue data presented in Appendix F (page F-43) indicate that "acephate residues ranged from 4.3 to 12.4 ppm on lettuce and broccoli measured 3 days after 2 lb/a.i./acre treatments" (emphasis added). "These residues degraded to 1.96 to 4.11 ppm 14 days after treatment. Such data indicate that initial insecticide residues could range from 10 to 50 ppm." Using these initial residue values, residue levels would be 8 to 20 percent after 14 days--not 33 to 46 percent. Further, these residue data are based on an initial application of 2 pounds of active ingredient per acre, or more than 2-1/2 times the amount of acephate applied per acre for gypsy moth control. Also see the responses to comments C30, C31, and C34.

C37 The rationale for determining the cancer risk for 4-chloroaniline (a breakdown product of diflubenzuron) is described in Appendix F (pages F-83 through F-84) and Appendix I (pages I-18 through I-21) and was further clarified in testimony presented before the District Court during OEC v. Kunzman (see Narrative Witness Statement of John Neisess, pages 14 to 16). The Court ruled that the FEIS did indeed discuss the cancer risks associated with exposure to diflubenzuron and discussed 4-chloroaniline in terms of its carcinogenicity (OEC v. Kunzman, page 661).

As discussed in Appendix I (page I-20), there are two theoretical pathways that the metabolic breakdown of diflubenzuron could take in soil, water, plants, and animals. These are discussed in the Diflubenzuron Decision Document (USEPA, 1979, as referenced in Appendix I). Only one of those two pathways could involve the formation of 4-chloroaniline at some point in the breakdown process. USDA searched the residue literature and found that 4-chloroaniline has rarely been found in nature, with the major exceptions being in fish and animals. Fish had the highest recorded residue levels of 4-chloroaniline (see page I-20).

Based upon recently completed cancer bioassays of diflubenzuron, EPA advised USDA that metabolites (such as 4-chloroaniline) do not pose a cancer risk to humans through dietary exposure (USEPA, 1985a, as referenced on page I-20). The reasoning behind this is that if 4-chloroaniline were produced by one of the breakdown pathways possible in plants, water, and animals, then the diet used in the rat and mouse cancer studies also would contain 4-chloroaniline.

As discussed in OEC v. Kunzman, USDA decided to take another look at the 4-chloroaniline issue based upon the NCAP comment letter (Letter 12) to the draft supplement to the FEIS. We concluded that where diflubenzuron was metabolized by an animal there might be a possibility of higher levels of 4-chloroaniline in animal tissue than would have resulted from the rat and mouse diets. The EPA data show that this is certainly true for fish. Therefore, the cancer analysis represents additional risks associated with the higher 4-chloroaniline residues that might form during metabolic breakdown in animals (fish) compared to the potential levels in the diet tested in the cancer bioassays. (See Narrative Witness Statement of John Neisess, pages 14 to 16.)

We recognize that diflubenzuron could concentrate in fish given its high octanol/water partition coefficient (see Appendix F, page F-39). However, the literature also shows that residue levels in fish tissue rapidly drop to undetectable levels once the exposure is removed, as would be the case in running water such as streams and rivers and in impoundments such as lakes and ponds where diflubenzuron rapidly binds with organic sediments. (See USEPA, 1979.) In addition, diflubenzuron is not expected to be found in large bodies of water such as lakes, ponds, and rivers because these larger bodies of water are avoided and do not receive direct diflubenzuron applications. Our primary concern is with the small, obscure streams and standing water bodies that cannot be seen from the air and that could receive a direct application. Also see response to comment C-70.

C38 See the responses to comments C27 and C28 for an explanation of why the 10-percent dermal absorption rate was used in the FEIS.

C39 The health risk analysis assumes that sensitive individuals are 100 times more sensitive to chemicals than the general population. Therefore, to account for possible adverse effects on sensitive individuals, the NOELs used in the analysis were first reduced by an arbitrary safety factor of 100; that is, they are 100 times lower than those used for the general public. Then margins of safety were calculated using these reduced NOELs. Because margins of safety must be 10 to 100 to ensure no adverse effects, the net effect is that we used a margin of safety of 10,000 for sensitive individuals relative to the original (unreduced) NOELs.

The discussion (in the Risk Evaluation section) of the potential health effects on sensitive individuals has been expanded to include specific effects for each chemical (see pages H-35 through H-38). In addition, a summary statement about possible effects to sensitive individuals has been added to the bulleted list on page H-2.

C40 The data used to generate the probability of aircraft spills were discussed on pages F-55 and F-56 of Appendix F of the FEIS. The data base covered the years 1958 to 1983 and pertained to spills occurring on spruce budworm spray projects because none had ever been recorded during this period on gypsy moth projects. When the current FEIS is updated, the 1985 Oregon project spills data will be taken into account and the probability of such spills will be recalculated.

C41 The section you refer to (pages H-30 to H-34) has been modified. Also, see our response to comment C7 for a discussion of how the ADI was used in the risk analysis.

C42 If one accepts EPA's conclusion that carbaryl does not cause birth defects in humans, then the statement as written in the Addendum is correct. We have rewritten the sentence to make that point clear. As discussed in the section on carbaryl's threshold effects, mixer/loaders and the general public exposed to worst case doses could be considered at risk if you rely solely on the lowest birth defect NOELs to determine risks (see page H-36).

C43 The statement you refer to (page H-33) has been modified to more accurately describe risks that could occur at dose levels below an ADI. Also, this section is only about threshold responses. Cancer and mutagenicity are discussed in the section about nonthreshold responses beginning on page H-38. No statements regarding "safe doses" of carcinogens or mutagens are made in this document.

C44 The purpose of Figure H-7 is to illustrate a general concept--margin of safety. This purpose would not be further served by adding other illustrations using different examples. If we were to use the examples requested in the comment, the new illustration would show the following: The worst case dietary dose of carbaryl (0.116 mg/kg/day) would be twice the kidney NOEL of 0.06 mg/kg/day, and it would be 27 times below the dog birth defect NOEL of 3.125 mg/kg/day. The new illustration also would show that the worst case dietary dose of carbaryl is just slightly above the ADI (0.1 mg/kg/day). All this information is contained in Tables 7 and 9 (on pages F-122 and F-124) and in Table I-1 (on page I-4).

C45 For the sake of readability, Appendix H contains no text references or list of citations. All source material is referenced in the FEIS and in Appendixes F and I. The text reference for the study in question can be found on page 45 of the FEIS, and the References Cited section clearly indicates that the study was prepared by the Chevron Chemical Company. Studies from research laboratories, universities, government agencies, and industry are all legitimate sources for information used in environmental impact statements.

C46 The study cited in comment C13 makes no mention of the use of antidotes in any of the poisoned individuals. See also the response to comment C13.

C47 The lowest carbaryl NOEL used in Appendix F is for birth defects in dogs (see Table 7, page F-122). We have modified the sentence (on page H-36 of the Final Addendum) to remind the reader that we are referring to the lowest carbaryl NOEL used in Appendix F. Also see the response to comments C6 and C15.

C48 EPA's official position concludes that carbaryl will not cause birth defects in humans (USEPA, 1984b, as referenced in Appendix I; and USEPA, 1980a, as referenced in Appendix F). Additional discussions on this point are presented on pages I-8 and I-9 of Appendix I, on page 57 of the FEIS, and on pages F-93 and F-94 of Appendix F.

C49 See change in text on page H-36.

C50 The discussion about birth defects on page H-36 has been modified in response to the comment. Regarding the first point, the worst case analysis assumes that sensitive individuals could be 100 times more sensitive to chemicals than the general population. This point is made on page F-99 in Appendix F (as well as on page H-33 and the response to comment C39). Regarding the second point, the lowest non-dog NOEL is 150 mg/kg/day and was derived from a study that used rabbits. If the dog NOEL for birth defects is used as a benchmark (that is, if it is assumed that humans respond to carbaryl the same way that dogs respond), then the margin of safety could be as low as 18 and the worst case carbaryl doses could be seen as leading to birth defects in the offspring of exposed individuals. But, again, it must be emphasized that there is doubt that carbaryl NOELs from dog studies can be applied to humans.

Further support for this conclusion is that EPA has concluded that carbaryl cannot cause birth defects in humans (USEPA, 1984b, as referenced in Appendix I).

C51 See the response to comment C40. The 1983 Willow Creek spill was part of the data base used in generating the spill probability figures.

C52 See the responses to comments C46 and C13.

C53 The cancer risks from realistic and worst case exposures are all given in Appendix F (pages F-74 through F-82) and in Appendix I (pages I-16 through I-26). Table H-7 includes only the weighted risks so readers would not be overwhelmed with numbers. The weighted risks were chosen because they give the most accurate picture of the cancer odds for all exposed individuals.

C54 See the response to comment C19. As discussed by Dr. John Neisess in OEC v. Kunzman (see Narrative Witness Statement of John Neisess, pages 11 to 13), USDA did not invalidate any cancer potency. His testimony identified that there was considerable uncertainty about the cancer potency of N-nitrosocarbaryl. It was pointed out that the Panel on Chemical Carcinogenesis Testing and Evaluation of the National Toxicology Program felt that the gavage method of testing (the method used for N-nitrosocarbaryl studies) may overestimate the incidence of cancer. See the response to comment C67 for a discussion on averaging cancer potencies.

C55 See the responses to comments C19 and C54.

C56 See the response to comment C37.

C57 See the responses to comments C35, C27, and C28.

C58 We disagree. We have presented the EPA official position of the Reproductive Assessment Group that concluded that carbaryl can be classified as a weak mutagen (USEPA, 1984b). This point is further discussed on page I-9 of the Addendum and on pages F-12 and F-13 of the FEIS.

C59 The text of Appendix I (page I-3) has been changed to reflect the different responses to acephate in various mutagenicity assays.

C60 We disagree. The study reported by Harry (1977) is discussed in Appendix I (page I-3) to provide descriptive information concerning the possible adverse toxic effects of carbaryl to humans. A single dose of 2.8 mg/kg is not considered to be a NOEL for carbaryl. The results also reported observations on a single test subject. NOELs are derived from well-designed, replicated studies.

C61 We disagree. The statement is an accurate discussion of the results of the Wills et al. (1968) study. The discussion in Appendix I is referring to the findings of the investigator under the conditions of the study. After administration of 0.13 mg/kg/day of carbaryl for 6 weeks, the authors of the study concluded that the decreased reabsorption capacity of the proximal tubules in the kidneys was reversible.

C62 The 1,166 mg/kg/day was the only dose administered during this study. The conclusions of the study were validated and reported by EPA (1984b).

C63 The study by Brookman et al., 1984, tested 42 "insecticides, solvents, emulsifiers, and mixtures thereof to determine whether these compounds are capable of enhancing the sensitivity of cultured mammalian cells to infection with vesicular stomatitis virus." The results show that none of the compounds significantly enhanced viral infection. These tests used the 1978 protocols of Rozee et al. described in Applied and Environmental Microbiology, volume 34, pages 297-300. The results show that three independent laboratories were unable to reproduce the viral enhancing ratios previously obtained by Rozee et al. This information, in addition to the study by Schmidt (1983) cited and discussed in Appendix I and to the testimony provided in the District Court during OEC v. Kunzman (see Narrative Witness Statement of Frank N. Dost, pages 6 and 7), satisfied USDA that carbaryl does not significantly enhance viral infections. This same conclusion has been added to Appendix I (pages I-10 and I-11).

Public involvement is discussed in the FEIS on pages 77 and 78. We believe that this involvement and notification process addresses the commenters concern about spraying uninformed people.

C64 See the response to comment C33.

C65 The discussion on page I-14 has been changed to clarify the fact that the 80 mg/kg dose was a single dose administered during the sensitive period in embryogenesis. Regarding the conclusions of Marsten et al. (1976) about the possible danger of trichlorfon, our discussions with the Environmental Protection Agency and Dr. Richard Thomas, a professional toxicologist, indicate that using the LOAEL and applying a safety factor of 2,500 is a commonly accepted practice.

C66 "Inconsistent" has been changed to "both negative and positive" (page I-15 of the Final Addendum).

C67 According to the publication "Quantitative approaches in use to assess cancer risk" by Elizabeth L. Anderson and the Carcinogen Assessment Group of the U.S. Environmental Protection Agency (Risk Analysis 3: 277-295, 1983), averaging is appropriate. The article proposed using a geometric mean. We chose to use a simple arithmetic mean because it is easier to understand. We also felt our cancer model already overestimated cancer potencies compared to other models. For example, we estimated the cancer potency for acephate to be 0.025 $(mg/kg/day)^{-1}$. The EPA comment letter (letter 19, comment d) informed us that they have now set the cancer potency for acephate at 0.009 $(mg/kg/day)^{-1}$.

C68 See the response to comment C67.

C69 Of the available studies that we reviewed, the actual presence of 4-chloroaniline was reported only in association with diflubenzuron breakdown in soil and metabolism in animals (especially fish). Our rationale for conducting the cancer risk analysis as we did is described in Appendix I (pages I-18 through I-21) and in testimony provided in the District Court during OEC v. Kunzman (see Narrative Witness Statement of John Neisess, pages 14-16). Also see our response to comment C37.

C70 The Diflubenzuron Decision Document (USEPA, 1979, as referenced in Appendix F and Appendix I) states on page 60 that "the likelihood of fish accumulating significant residues of diflubenzuron is thought to be quite low. It is possible that, through runoff, drift, or a combination thereof, water concentrations of diflubenzuron as high as 0.001 ppm might, on occasion, be sustained over a period of at least a few days. If such were the case, residues in fish meat might reach levels approaching 0.05 ppm, assuming a biomagnification of 50 times (Aperson et al. 1978, Booth et al., 1976, Schaefer et al., 1978). Fish are also capable of rapidly depleting residues of diflubenzuron from the body once exposure to the compound is terminated (Booth et al., 1976, Schaefer et al., 1978)" (emphasis added). Based on this information, EPA concluded that "the potential for significant human dietary exposure to diflubenzuron via residues in fish would appear to be slight" (emphasis added). (USEPA, 1979)

In the gypsy moth risk analysis, we use a bioconcentration factor of 1 based upon the rationale described in Appendix F (page F-39). The residue levels in fish meat described on page F-39 are estimated to range from 0.05 ppm to 0.71 ppm, which are the same values for diflubenzuron in fish tissue that EPA cites in the Decision Document. In addition, the worst case residue value assigned to fish meat in the risk analysis, and subsequently used to calculate cancer risk (0.71 ppm), is 14 times greater than EPA's high estimate.

On page 61 in the Diflubenzuron Decision Document, EPA estimates the "worst case" total dietary exposure to be 3.4×10^{-6} mg/kg. The realistic and worst case dietary doses that are calculated using the methods and assumptions in the gypsy moth risk analysis (such as a bioconcentration factor of 1.0) are 200 and 2,500 times, respectively, greater than the worst case total dietary exposure described by EPA in the Diflubenzuron Decision Document. Therefore, we do not believe that we have underestimated exposure or risk from exposure to diflubenzuron or the metabolite 4-chloroaniline.

C71 Information regarding the APHIS projects has been included in the discussion on page I-23.

C72 No comment C72 provided.

C73 Careful review of Appendix E shows that two, not three, applications of carbaryl in gypsy moth eradication projects are most common. As indicated in Appendix E, the State of California commonly uses three applications, but these projects are not cooperatively funded by USDA. Therefore, we felt that it was more appropriate to discuss cumulative effects in terms of situations common to our (USDA) programs--that is, two applications. In the event that three applications are planned in some future USDA project, then the specific cumulative effects will need to be addressed in site-specific environmental documents by using the procedures and methods described in the risk analysis (pages F-82 and F-83).

C74 We fail to understand why you believe the sentence quoted is misleading. The specific persistence of each chemical is not what is important in this statement. The point as stated is that some degradation will have occurred during the time prior to the second application. Therefore, the total dose will be some amount less than two realistic case doses.

C75 See correction in Appendix I on pages I-23 and I-24.

C76 The ADI for carbaryl used in the FEIS and the Addendum was set by EPA. The statement referred to in the comment (pages I-23 and I-24) is correct. Also, see the response to comment C73 regarding three applications of carbaryl.

C77 The sentence indicates that, if people can avoid dietary exposure, health risks should be low even when exposed to a double application.

C78 Residue data from grass, shrubs, and trees were used to determine the insecticide dose received by animals rabbits or goats). For humans, residue data from vegetable crops were used to calculate the dose received from the consumption of such products. These latter data were used instead of residue data on grass and shrubs because they represent the type of food humans normally consume. (Also see the response to comment C34.) The acephate reference was missing. The Chevron, 1973, reference has been added to clarify the void. This was the reference for the data discussed and referenced on page F-43. Also see the response to Letter 12, comment J, page G-9 of the FEIS. In addition, this point was ruled on in OEC v. Kunzman. The Court indicated that USDA's choice of studies upon which to rely was within its discretion (OEC v. Kunzman, page 662).

C79 Insecticide persistence was previously addressed in the FEIS in response to Letter 12, comment K, page G-9, and again in the narrative statement of Dr. John Neisess in OEC v. Kunzman (see Narrative Witness Statement of John Neisess, page 8). It also was ruled on in the opinion issued by the District Court as discussed in response to comment C78.

C80 The issue of insecticide residues and persistence in water was discussed in the narrative statement of Dr. John Neisess in OEC v. Kunzman (see Narrative Witness Statement of John Neisess, pages 6 to 8) as well as on page I-26. (Also see response to comment C78).

h We disagree that the EIS should be rewritten because of the comments mentioned. We have addressed each comment indicating what studies or scientific data were used in the FEIS or Addendum and why they were used. Furthermore, in its opinion the U.S. District Court held that the FEIS was legally adequate (OEC v. Kunzman, page 666) with the exception of Appendix F. The Court held that, although Appendix F of the FEIS contains all necessary information, it was too technical, complex, and full of equations and calculations (OEC v. Kunzman, page 665). Appendix H presented in the Final Addendum specifically addresses the Court's concern regarding the clarity of technical data used in the worst case analysis.

Since the concerns of the Court and all commenters have been addressed adequately, we believe there is no reason to rewrite the FEIS. See also the response to comment C19.

i We disagree. The point about the organization of the FEIS as supplemented and the Addendum already has been discussed in our response to comment a in this letter.

j We agree that many of the comments presented here are familiar. The majority were addressed by USDA in response to comments on the Draft EIS and printed in the FEIS as Supplemented 1985. They were raised by the plaintiffs during the OEC v. Kunzman trial and addressed by government briefs as well as defense witnesses. Finally, they were ruled upon by the U.S. District Court (OEC v. Kunzman, page 662). The only issue decided in favor of the plaintiffs involved the readability of Appendix F concerning the Analysis of Human Health Risks of Using Acephate, Carbaryl, Diflubenzuron, and Trichlorfon in Gypsy Moth Suppression and Eradication Projects. In response to the U.S. District Court's direction, USDA has issued a plain language version of Appendix F as an Addendum to the FEIS as Supplemented 1985 that informs the public and decision-makers of the potential health risks of the insecticides.

k Because Attachment 1 does not specifically address the Draft Addendum, we believe that it is unnecessary to respond to this attachment in this forum. The proper forum to address the Appellate Brief included as Attachment 1 is a court of law. Any response to the Appellate Brief will be made by USDA's statutorily appointed legal representative, the Department of Justice.

l Attachment 2 also does not specifically address the Draft Addendum. Dr. Mary O'Brien's narrative statement was written in the course of the OEC v. Kunzman litigation—that is, before the Draft Addendum was written. USDA's response to this statement can be found in the documents and testimony presented to the U.S. District Court during that litigation. Judge Redden's Opinion in OEC v. Kunzman also may be of assistance to anyone wishing to review or evaluate Attachment 2. (Judge Redden's Opinion can be found on pages J-138 to J-142).

Letter 17 No response required.

Letter 18 No response required.

Letter 19

a Your requested change has been made on page H-1.

b See the change in the discussion on page H-3.

c Your suggested change has been made on page H-11.

d Comment noted. This change will be incorporated the next time the FEIS is revised. The agencies will continue to monitor the status of EPA peer group classification of acephate and all other chemicals involved in this program.

e Your suggested change has been made on page H-11.

f A surrogate chemical was not used to develop the cancer potency for trichlorfon. As discussed on page F-19 of Appendix F, the potency figure is based on data reported by Machemer (1981). To obtain a positive slope, the higher level of malignant tumors in the untreated control group was ignored and the response curve was forced through zero.

g See the change in the discussion on page H-15.

h See the change in the discussion on page H-13.

Letter 20

a We agree that available evidence suggests that carbaryl does not cause cancer. The issue is whether N-nitrosocarbaryl, a carcinogen, can form in the human stomach as a result of exposure to carbaryl. Because the formation of N-nitrosocarbaryl is uncertain, the "worst case" process requires that the worst be assumed--namely, that exposure to carbaryl can lead to cancer. This is explained on pages H-3 and H-4, F-14 and F-15, I-11 and I-12, and pages 38 through 42 of the FEIS.

b Your statement that Market Basket Surveys have detected minute levels of carbaryl in foodstuffs supports the statement on page H-4 of the Final Addendum.

c The "worst case" process requires that when data are lacking or there is uncertainty, the worst case must be assumed. This is explained on pages H-3 and H-4. The data on N-nitrosocarbaryl analyzed in Appendixes F, H, and I agree with the conclusions drawn by Richard and Dorough (1984). Even under worst case assumptions, the risk of cancer from N-nitrosocarbaryl is very small.

d See the response to comment C50, Letter 16.

e This was a typographical error. The correction has been made in Table H-7 in the Final Addendum.

Letter 21

a No response required.

b No response required.

Letter 22

a As indicated in the Introduction on page vii, Appendix H is intended to be a plain language version of Appendix F. It presents in clear language the risks to human health of using four insecticides to control gypsy moths. Where there are risks to health, specific symptoms are presented in the Risk Evaluation section. These risks also are summarized in the Overview section on pages H-1 and H-2.

b The format for the Addendum was chosen because it was important to ensure that there was some continuity throughout the entire FEIS. This format is the same basic format used for the Final Environmental Impact Statement, issued on March 8, 1985, of which this document is a part. We have tried to better differentiate sections by typesetting the heads for the four major sections--Overview, Hazard Identification, Exposure Analysis, and Risk Evaluation--as well as for their subsections, and by using boldface for the next level of heads. The Table of Contents indicates the relative importance of headings. We think this helps the reader to distinguish clearly where sections begin and end.

c A reference to Table H-5 was added to the paragraph as you suggested (see page H-28). Pages F-75 and F-76 in Appendix F explain in detail how average lifetime doses were calculated for N-nitrosocarbaryl.

d Explanations of calculations, as well as references to pages in Appendixes F and I that show how to calculate lifetime doses have been added to the text (see page H-28). We did not include actual examples of the calculations because these calculations are shown quite clearly in Appendix F (page F-80) and Appendix I (pages I-20 and I-21) and because such calculations would hamper readability in this appendix.

e This point was made on page H-29 of the Draft Addendum and remains on page H-30 of the Final Addendum. We also have added a note to the table (now table H-6) to further aid the reader.

f We intentionally avoided repeating specific exposure and threshold levels in this section so the reader would not be overwhelmed with data and so we could get right to the "bottom line" of the risk analysis: where the estimated doses stand in relationship to the toxicity levels. The numerical comparisons referred to in the comment are the margins of safety and are described in Figure H-7. They also are presented in Tables 8 through 15 on pages F-123 through F-130 in Appendix F; we have added a reference in Appendix H to those tables. We also have added a sentence to the caption of Figure H-7 indicating that the margins of safety are derived by comparing the estimated doses shown in Tables H-4 and H-5 (now tables H-2 and H-3) with the NOELs shown in Table H-1.

g Tables 1 through 4 and Table 7 in Appendix F list the birth defect NOELs for the four chemicals. We have added the lowest birth defect NOEL for each chemical to Table H-1.

Letter 23 Comment noted. See response to comment C43.

OEC v. Kunzman
Opinion

OREGON ENVIRONMENTAL COUNCIL; Citizens For the Safe Control of the Gypsy Moth; Elaine Olsen and Glen Olsen, Plaintiffs,

and

Friends of the Earth; National Coalition Against the Misuse of Pesticides, Plaintiffs/Intervenors,

v.

Leonard KUNZMAN, Director, State of Oregon, Department of Agriculture, State of Oregon, Department of Agriculture; United States Department of Agriculture; John R. Block, Secretary, United States Department of Agriculture; et al., Defendants.

and

Oregonians For Food and Shelter, Inc., Defendants/Intervenors.

Civ. No. 82–504–RE.

United States District Court,
D. Oregon.

April 26, 1985.

Suit was commenced seeking to enjoin spraying program designed to eradicate gypsy moths in an urban area. After remand, 714 F.2d 901, the District Court, Redden, J., held that while main text of Department of Agriculture's final environmental impact statement concerning spraying program designed to eradicate gypsy moths in an urban area was legally sufficient, in view of legal insufficiency of worst case analysis, which failed to meet requirements of applicable regulation in that it was hypertechnical, complex, and replete with lengthy equations and calculations, Department would be enjoined from conducting its proposed spraying program.

Order accordingly.

1. Health and Environment ⇔25.15(3.2)

When a final environmental impact statement is filed it is subject to judicial

review for compliance with National Environmental Policy Act. National Environmental Policy Act of 1969, § 102, 42 U.S.C.A. § 4332.

2. Health and Environment ⇔25.15(10)

Neither National Environmental Policy Act [42 U.S.C.A. § 4332] nor its legislative history allows a court to substitute its judgment for that of preparing agency as to environmental consequences of proposed actions. National Environmental Policy Act of 1969, § 102, 42 U.S.C.A. § 4332.

3. Health and Environment ⇔25.15(2)

While main text of Department of Agriculture's final environmental impact statement concerning spraying program designed to eradicate gypsy moths in an urban area was legally sufficient, in view of legal insufficiency of worst case analysis, which failed to meet requirements of applicable regulation in that it was hypertechnical, complex, and replete with lengthy equations and calculations, Department would be enjoined from conducting its proposed spraying program.

Larry N. Sokol, Jolles, Sokol & Bernstein, P.C., Portland, Or., John E. Bonine, Michael D. Axline, Ralph Bradley, Bradley & Gordon, Eugene, Or., for plaintiffs and plaintiffs/intervenors.

Charles H. Turner, U.S. Atty., Thomas C. Lee, Asst. U.S. Atty., Portland, Or., Dorothy R. Burakreis, R.W. Rodrigues, Elizabeth Ann Peterson, Land and Natural Resources Div., General Litigation Section, U.S. Dept. of Justice, Washington, D.C., John DiLorenzo, Jr., Brendan Stocklin-Enright, DiLorenzo & Dietz, Portland, Or., for defendants and defendants/intervenors.

OPINION

REDDEN, District Judge:

BACKGROUND

Plaintiffs commenced this suit in Spring of 1982 seeking to enjoin the spraying of carbaryl over 6,400 acres in Salem, Oregon. The spraying was the crux of a program designed to eradicate gypsy moths discovered in that urban area. Plaintiffs alleged that the spray program violated the National Environmental Policy Act (NEPA), 42 U.S.C. § 4332 (1976), the Administrative Procedure Act (APA), 5 U.S.C. §§ 702 and 706 (1976) and several other statutes or regulations.

After a trial on the merits I determined that the federal defendants had not violated NEPA. On appeal, the Ninth Circuit determined that the federal defendants had in fact violated NEPA by failing to prepare adequate site specific environmental statements. *Oregon Environmental Council v. Kunzman*, 714 F.2d 901, 905 (9th Cir. 1983).

On January 26, 1984, I issued a permanent injunction prohibiting federal defendants from implementing any program for aerial broadcast spraying of carbaryl in populated areas in Oregon until they prepared a legally adequate Environmental Impact Statement (EIS). On March 16, 1984, federal defendants issued a new Programmatic Environmental Impact Statement (PEIS) for gypsy moth eradication and suppression programs in the United States. Plaintiffs challenged the PEIS by filing a motion for a temporary restraining order, preliminary injunction and an order to show cause to prevent federal defendants from aerial broadcast spraying anywhere in the United States pursuant to the March 16, 1984 PEIS. Plaintiffs alleged that the PEIS was legally deficient in a number of ways.

On May 3, 1984, Friends of the Earth, National Coalition Against the Misuse of Pesticides moved the court for an order allowing it to intervene in plaintiffs' motion. I granted the motion on the same day. Following a number of postponement requests by both sides, a final hearing date was set for September 25, 1984. A pretrial conference was scheduled for August 31, 1984.

On August 20, 1984, the government withdrew the contested PEIS, issuing its intent to supplement it. At the pretrial conference the government informed the court that it would have a draft supplement for public review by November 1, 1984. The draft supplement was not made available to the public, however, until December 29, 1984. A 45 day comment period followed the issuance, after which the government began preparation of the final EIS as supplemented.

On March 1, 1985, Oregonians for Food and Shelter, Inc. moved the court for an order granting it permission to intervene on behalf of defendants. That motion was granted on March 29, 1985. Meanwhile, the final Environmental Impact Statement (FEIS) as Supplemented (FEIS), for the eradication and suppression of gypsy moths was filed on March 23, 1985.

Plaintiffs challenged the adequacy of the FEIS on numerous grounds and a trial on the merits was held April 16–18, 1985. For the reasons set forth below I enjoin the use of carbaryl, trichlorofon, acephate and diflubenzuron in Oregon effective this date, and enjoin the use of the same synthetic chemical sprays elsewhere in the United States effective January 1, 1986. The use of Bacillus thuringiensis (B.t.) is not enjoined in Oregon or elsewhere in any program based upon the current Final Environmental Impact Statement as Supplemented, 1985.

At trial plaintiffs alleged that they were precluded from commenting on the FEIS's worst case analysis (WCA) because it was "tacked on" to the March 1984 PEIS at the last minute. Plaintiffs further challenged the WCA on the grounds that it did not mention that diflubenzuron might cause cancer, or that children and chemically sensitive persons would be adversely affected by the use of the synthetic pesticides discussed in the FEIS. Plaintiffs also contended that the WCA did not consider the expected synergistic or cumulative effects of the synthetic pesticides and other toxic agents in the environment. Additionally, plaintiffs alleged, the WCA underestimates the period the synthetic pesticides remain in the environment and their absorption rates.

Plaintiffs also criticized the use of acceptable daily intake (ADI) and no observable effect level (NOEL) figures in computing health risks to humans. Plaintiffs also argued that defendants failed to fill "data gaps" as statutorily required and that the alternatives to synthetic pesticides were buried in the body of the FEIS. Plaintiffs also challenged the FEIS on the ground that it does not give a total estimate of the number of people that might develop cancer as a result of spraying the synthetic pesticides discussed. Finally, plaintiffs contend that the FEIS fails to meet the regulatory requirements of brevity and clarity.

The PEIS was withdrawn in August 1984 and from then to the present, federal defendants have contended that the court lacks jurisdiction to review the EIS in any form because there is no final agency action.

A. *NEPA's Requirements and Purpose*

Section 102(2)(c) of NEPA requires the preparation of an EIS whenever "major Federal actions significantly affecting the quality of the human environment" are undertaken. Clauses (i) through (v) specify the content of the EIS and establish the adequacy requirement. These requirements are expanded and defined by regulations promulgated by the Council on Environmental Quality (CEQ). The CEQ is an agency established by Title II of NEPA for the purpose of promulgating advisory rules and regulations and to review and evaluate federal programs.

Although the CEQ is advisory in nature, federal courts have accorded it great deference. *Environmental Defense Fund, Inc. v. Hoffman*, 566 F.2d 1060 (8th Cir.1977); *Silentman v. Federal Power Commission*, 566 F.2d 237 (D.C.Cir.1977); *State of Alaska v. Carter*, 462 F.Supp. 1155 (D.Alaska 1978).

The CEQ regulations state that the purpose of an EIS is to serve as an action forcing device to insure that the policies and goals of the Act are infused into ongoing and proposed federal programs. An EIS is to provide a "full and fair discussion of significant environmental impacts" and inform the public, as well as decisionmakers, of available alternatives which would avoid or minimize adverse effects. An EIS is to inform the public and decisionmakers, in clear and succinct language, of proposed federal action, the harms and health risks associated with the proposed actions, and any reasonable alternatives.

B. *Standard of Review*

[1] NEPA does not expressly provide for judicial review of agency actions but the APA does. Also, courts have uniformly held that judicial review of agency decisions is implied by NEPA. *Calvert Cliffs Coordinating Committee, Inc. v. United States Atomic Energy Commission*, 449 F.2d 1109 (D.C.Cir.1971); *Environmental Defense Fund, Inc. v. Corps of Engineers of the United States Army*, 470 F.2d 289 (8th Cir.1972), *cert. denied*, 412 U.S. 931, 93 S.Ct. 2749, 37 L.Ed.2d 160 (1973). When a FEIS is filed it is subject to judicial review for compliance with NEPA.

The adequacy of an EIS depends upon whether it was prepared in observance of the procedures required by NEPA, 5 U.S.C. § 706(2)(D). *See also, Lathan v. Brinegar*, 506 F.2d 677, 693 (9th Cir.1974) (en banc). Under this standard of review, courts employ a "rule of reason" that inquires whether an EIS contains a reasonably thorough discussion of the significant aspects of the probable environmental consequences. *Trout Unlimited, Inc. v. Morton*, 509 F.2d 1276, 1283 (9th Cir.1974). Although this is a flexible standard not readily susceptible to a precise definition, several courts have attempted to explain it.

In *Silva v. Lynn II*, 482 F.2d 1282 (1st Cir.1973), the court held that the impact statement must allow the court to determine whether the agency made a "good faith effort" to take environmental values into account. To meet this requirement the impact statement must fully explain its inquiry, analysis and reasoning.

The Court in *Warm Springs Dam Task Force v. Gribble*, 565 F.2d 549 (9th Cir. 1977), requires the reviewing court to make a pragmatic judgment as to whether the EIS's form, content and preparation fostered both informed decisionmaking and informed public participation.

The Second Circuit explains these two approaches in *Sierra Club v. United States Army Corps of Engineers*, 701 F.2d 1011, 1029 (2nd Cir.1983). The Court said:

> [t]he ... [impact statement] must set forth sufficient information for the general public to make an informed evaluation, ... and for the decisionmaker to consider fully the environmental factors involved and to make a reasoned decision after balancing the risks of harm to the environment against the benefits to be derived from the proposed action ... [the impact statement gives] assurance that the stubborn problems or serious criticisms have not been swept under the rug.

The courts usually refer to the duty to prepare an adequate impact statement as the agency's "procedural" duty to comply with NEPA. The agency must comply with NEPA's procedures to insure the EIS contains an adequate analysis for the public and decisionmakers.

I am limited to reviewing the agency's procedural duty and may not substitute my judgment for that of the agency concerning the wisdom or prudence of the proposed programs. *California v. Block*, 690 F.2d 753, 761 (9th Cir.1982). Once satisfied that the agency has taken a "hard look" at the environmental consequences of the programs, I end my review. *Kleppe v. Sierra Club*, 427 U.S. 390, 410 n. 21, 96 S.Ct. 2718, 2730 n. 21, 49 L.Ed.2d 576 (1976).

C. *Jurisdiction*

Federal defendants assert that this court lacks jurisdiction to review the FEIS because there is no final agency action. Defendants argue that the FEIS was noticed in the Federal Register on March 15, 1985, and that there was no final agency action until 30 days after that pursuant to 40 C.F.R. § 1506.10. The trial on the adequacy of the FEIS commenced on April 16, 1985, more than 30 days after the FEIS was noticed in the Federal Register. Moreover, 40 C.F.R. § 1500.3 specifically states:

> It is the Council's intention that judicial review of agency compliance with these regulations not occur before an agency has *filed* the final environmental impact statement....

(Emphasis added).

The FEIS was filed on March 23, 1985. Thus, this court has jurisdiction over this case as the agency's action was final as of the date of the commencement of the trial.

D. *Plaintiffs' Substantive Challenges*

1. *No Comment Period for WCA*

Plaintiffs contend that the federal defendants "tacked on" the WCA at the last minute, precluding any public comment on it. This argument is without merit. The PEIS, including its WCA, was filed on March 23, 1984 and was subject to a 45 day public comment period which expired on May 7, 1984. 49 Federal Register 10,953.

Plaintiffs fail to state why they were precluded from commenting on the WCA during this 45 day period. Further, the draft supplement to the PEIS, released on December 29, 1984 subject to a 45 day comment period, also contained a WCA. I find that plaintiffs were given opportunity to comment. The plaintiffs do not prevail on this issue.

2. *Carcinogenicity of Diflubenzuron*

Plaintiffs contend that the federal defendants ignored the fact that the EPA has determined that 4-chloroaniline, a metabolite of diflubenzuron, is carcinogenic. Plaintiffs say that the omission of this discussion is misleading and renders the FEIS legally inadequate. Plaintiffs' contention is unfounded for two reasons.

First, the FEIS contains a discussion of the cancer risks associated with the ingestion of products which have been derived from animals exposed to diflubenzuron and thus contain various amounts of 4-chloroaniline. 64, F-12, F-21, F-83, F-84, G-21 at comment response kk. Diflubenzuron itself is noncarcinogenic.

Secondly, as was discussed by expert witnesses at trial, there is debate in the scientific community over whether 4-chloroaniline is in fact carcinogenic. The FEIS discusses 4-chloronailine in terms of its carcinogenicity. The plaintiffs' complaint that the FEIS does not alert the reader to the *known* carcinogenic risks associated with the use of diflubenzuron (through the ingestion of its metabolite 4-chloroaniline) stretches the disclosure requirements of NEPA too far.

3. *Children and Chemically Sensitive Individuals*

Plaintiffs argue that the FEIS fails to adequately discuss the risks of exposure to the synthetic pesticides to children and chemically sensitive individuals. Plaintiffs acknowledge that the FEIS includes a safety factor of ten to account for chemically sensitive persons, but argue that this factor is too low because such persons can be hundreds or thousands of times more sensitive than the general population.

A discussion of adverse health effects on chemically sensitive persons is included in Appendix F (WCA) of the FEIS at pages F-98–F-100. The safety factors utilized in that discussion look into many biological factors such as age (young children and the elderly), sex, genetic composition and preexisting illness. Page F-99 states

> In order to account for possible impacts to sensitive individuals, the NOELS were reduced by an arbitrary safety factor of 100. ADIs already include a safety factor (100 in most cases) which was incorporated in part, to account for differential responses within the population. Although 100 is arbitrary it is based on a review of selected literature on variable human responses to foreign chemicals (xenobiotics) or diseases. (Citation omitted). Depending on the specific substance or disease, there was a 3.7–100 fold variation in response. However, 80–95% of the variation fell within a 10 fold factor.

At trial testimony was that the 5–20% of the sensitive population not falling within the ten fold factor fell on both sides of that

factor. Thus, not all persons outside of the ten fold factor had greater sensitivity; some had less. Also, there was no testimony or evidence that sensitive individuals exceed a 100 fold factor. Accordingly, the FEIS adequately discussed the potential risks to chemically sensitive persons, and plaintiffs fail in this contention.

4. *Synergistic and Cumulative Effects*

Plaintiffs claim the FEIS should not have attempted to discuss the synergistic effects of the four proposed synthetic pesticides in general terms because the actual effects are not yet known. Plaintiffs suggest that the FEIS should have concluded that "we do not know what health effects could occur as a result of synergism of these pesticides with other chemicals but will assume that such effects will occur in the worst case." Plaintiffs' trial memorandum, page 15.

[2] The FEIS, as acknowledged by plaintiffs, contains a discussion of synergism at pages F-101—F-104. The FEIS discloses the fact that the actual synergistic effects of the synthetic pesticides with other chemicals are unknown and declines to suppose an effect. This is all that is required. Moreover, neither the statute nor its legislative history allows a court to substitute its judgment for that of the preparing agency as to the environmental consequences of the proposed actions. *Kleppe v. Sierra Club*, 427 U.S. at 421, 96 S.Ct. at 2735.

Plaintiffs further argue that the FEIS seriously underestimates the cumulative effects of repeated exposures to the proposed pesticides. At page F-103 of the FEIS there is a discussion of the cumulative effects of eating foods receiving multiple sprayings of the synthetic pesticides within a single season. Plaintiffs say this is inadequate because it does not take multiple direct exposures into consideration. At trial Dr. Schneeberger testified that the discussion of cumulative effects took into consideration not only the effects of eating foods which had been sprayed more than once, but also multiple direct exposures to the individual as well. Thus direct exposure was taken into consideration and the document is adequate in discussing the cumulative exposure effects. Plaintiffs do not prevail on these issues.

5. *Absorption and Persistence Rates*

Plaintiffs argue that the calculations in the FEIS of the persistence and absorption of acephate on vegetables are overly optimistic. Page F-43 contains these calculations. Plaintiffs point to the estimate that only five percent of acephate residues will wash off vegetables after one day, due to absorption of the chemical. Plaintiffs agree with this estimate and state that the calculation at F-43 amounts to a 77% wash off of residues. In reaching this conclusion plaintiffs assumed that vegetables would have a residue of 50 parts per million, while the FEIS stated that only 4.0 to 11.4 parts per million remained after washing. At trial it was determined that vegetables will retain 4–15 parts per million of acephate and that the numbers in the FEIS were correct. (The 50 parts per million figure related to carbaryl residues, not acephate residues). This argument also fails, and I find the FEIS adequate on that issue.

Plaintiffs also argue that certain studies indicate that diflubenzuron and acephate may take considerably longer than the 14 days to degrade, the figure used by the FEIS. At trial defense witnesses explained why they did not rely on those studies in preparing the FEIS. They stated that the studies had been severely criticized because they had considered excessive use of the pesticides, and killed soil microorganisms necessary for the degradation process, or because the study conditions had been in controlled environments not representative of the actual environment.

I find that the agency's choice of studies on which to rely is within its discretion. I am precluded from reviewing such decisions unless I find them arbitrary or capricious. 5 U.S.C. § 706(2)(A). I find this decision to be reasonable, and plaintiffs fail on this issue.

6. *Use of ADIs and NOELs*

Plaintiffs contend that the FEIS stresses that NOEL and ADI figures indicate levels of safety and represent no risk situations for the environment and human health. At trial there was lengthy discussion about the use of these figures in assessing human health risks. These numbers are derived from animal studies and are therefore not truly correlative to human risks.

The FEIS defines a NOEL as the level of exposure of an experimental organism to a chemical, at which no adverse effects are observed. This figure does not mean that no adverse effects actually occur at that level, only that none are observed.

ADIs are defined in the FEIS as the maximum dose of a substance that is to be anticipated to be without lifetime risk to human health when taken daily. ADIs are established by dividing NOELs by a factor of ten to account for increased sensitivity of humans over the test animals used to establish the NOELs. These figures are then reduced another ten times to account for more sensitive humans. Thus, ADIs are typically 100 times lower than NOELs. At trial Drs. Richard Niesess and Richard Thomas testified that these values are routinely used in assessing human risks to chemical exposure and other environmental contaminants. No values are available for humans, because no medical or scientific research of this type has been done using humans as experimental models. Moreover, there was testimony that the use of these figures tend to overestimate associated risks to humans. Thus, the use of the figures to estimate human health risks is reasonable and does not render the document inadequate. Plaintiffs cannot prevail on this issue.

7. *Data Gaps*

Plaintiffs argue that defendants failed to conduct independent research to fill informational gaps as to negative effects of the proposed chemical sprays. 40 C.F.R. § 1502.22 provides that in evaluating adverse effects on the human environment where there are gaps in relevant information or scientific data, the agency must make it clear that such information is lacking, or that uncertainty exists. If the information relevant to adverse impacts is essential but not known, the agency must fill that gap by independent research. *Save Our Ecosystems v. Clark*, 747 F.2d 1240, 1249 (9th Cir.1984). An agency need not do independent research if the costs of doing the research are exhorbitant or the research means are beyond the state of the art. 40 C.F.R. 1502.22.

Plaintiffs argue that defendants have ignored this mandate by providing no basis for measuring the costs of research, ignoring human health risks in considering what costs are exhorbitant, and claiming resolution of scientific uncertainty is beyond the state of the art.

The FEIS contains extensive bibliographies at pages 85–97 and F–105–F–113. Page F–7 lists numerous data bases which were searched by the agency in gathering information for the preparation of the FEIS. Several witnesses testified at trial that there are "data gaps" in the relevant information. The research needed to fill these gaps would take many years and require great expense to complete. Scientific studies, in order to be accurate and trustworthy, must be painstakingly performed, repeated and peer reviewed. This process takes many years to complete.

The data on the possible human health effects of these synthetic pesticides were not available to the agency, and could not have been obtained by it either through literature searches or independent research. Accordingly, it was reasonable for the agency to prepare a worst case analysis.

Plaintiffs suggest that the agency compute the total health care expenditures of all humans adversely effected by the programs instituted and compare that figure with the amount of money the government will expend to implement the programs, in order to determine if the costs of research are exhorbitant. This suggestion is illogical. Plaintiffs would require the agency to determine health care costs for health effects presently unknown. That is, the agency would have to have available to it all the scientific data that plaintiffs insist it must research. Plaintiffs do not prevail on this issue.

8. *Alternatives Buried*

Plaintiffs contend that alternatives to chemical spraying have been buried in the body of the FEIS and thus suggest to decisionmakers that none exist. This challenge to the adequacy of the FEIS is without merit. Alternatives to chemical spraying are discussed in the FEIS at pages ii, 9, 14, 15, 16, 20–24.

Other areas of controversy are also discussed throughout the document despite plaintiffs' contention that the worst case issues of cancer, mutations and birth defects are buried. The summary clearly identifies major concerns and issues associated with the proposed spraying programs. The first issue mentioned in the summary is human health. Also mentioned are the need for public involvement and education, environmental effects of certain insecticides and the need for the development of alternative suppression methods not involving chemical spraying. Both the summary and the text of the FEIS identify each of the four proposed synthetic insecticides and the cancer risks based on an individual's exposure to these compounds. Pages vi, vii, 58, 61, 64, 67, table 17, page F–133. *See also* tables 8–11, pages F–123–126. I do not agree that these alternatives are buried and that the FEIS is therefore invalid.

9. *No Estimation of Total Cancer Victims*

Plaintiffs assert that the FEIS must contain an estimate of the total number of persons expected to develop cancer as a result of the proposed spraying programs. Plaintiffs state that the FEIS contains no such estimate and is therefore illegally inadequate. Plaintiffs cite no authority for this proposition, but argue that, in their opinion, such an estimated figure is necessary. I do not find the FEIS inadequate for this reason. On pages F–95 and F–96 the FEIS discusses the estimated incidences of cancer assuming that all people living in a treated area will receive the highest estimated exposure. These figures were obtained by using data collected during the previous four years of spraying for gypsy moth suppression and eradication. Pages F–76 through F–84 contain the formulas and equations necessary for calculating this risk. These equations take into account exposure through spraying, dietary sources (both from insecticide on food directly, and from insecticide ingested by animals which are later eaten by humans), watershed, soil concentration and worker exposure. The equations also consider double exposure risks, i.e., direct exposure from spraying or working and dietary exposure.

Plaintiffs' argument that the FEIS must go beyond these calculations and set an overall specific estimated number of cancer victims is unreasonable for two reasons. First, the FEIS is intended to outline the risks associated with the particular chemicals. The FEIS is then to be supplemented by environmental assessments (EAs) from the individual state agencies that intend to use the chemicals for gypsy moth suppression. The EAs are to correlate the risks set out in the FEIS to the specific areas to be sprayed in the particular state. The FEIS cannot estimate the total number of human exposures without the EAs from the particular states. Any overall number otherwise reported would be conjectural.

Secondly, the FEIS clearly states that 14 states anticipate participation in the federal spraying program. It is unreasonable to require a figure of cancer incidences on a *nationwide* basis. "Common sense ... teaches us that the 'detailed statement' ... cannot be found wanting simply because the agency failed to include every alternative device and thought conceivable by the mind of man." *Vermont Yankee Nuclear Power Corp. v. Natural Resources Defense Council, Inc.*, 435 U.S. 519, 551, 98 S.Ct. 1197, 1215, 55 L.Ed.2d 460 (1978).

10. *Clarity of FEIS*

40 C.F.R. §§ 1502.1 and 1502.2(b) state that the information in an EIS must be

"concise, clear and to the point" and that statements should be "analytic rather than encyclopedic." Section 1502.8 further states that the document should be written in "plain language so that decisionmakers and the public can *readily understand them.*" (Emphasis added). Plaintiffs assert that the FEIS is highly technical, esoteric and incomprehensible not only to the general public and majority of decisionmakers, but also to readers with advanced scientific backgrounds. I agree with plaintiffs that the WCA does not meet the regulatory mandate of clarity.

A thorough search of the case law and relevant literature has failed to produce any precedent or discussion regarding the "readability" of an EIS. Mention has been made in a few cases of the requirement of understandability in § 1502.8. However, no court has ever invalidated an EIS on the grounds that the document was not "readily understand[able]" by the public and decisionmakers.

I find that the text of the FEIS (pages i–78), although technical does meet the requirements of § 1502.8. That portion of the document discusses six alternatives for the control of the gypsy moth: the four synthetic pesticides, B.t. and integrated pest management. The environmental consequences of each are discussed in clear language. Full disclosure of the consequences of each alternative is in the text. Also included is mention of the fact that more research is needed on the human health risks posed by the use of the four synthetic pesticides.

As stated, because relevant information on the health risks of these pesticides is lacking, the agency is required to include a "worst case analysis" in the FEIS pursuant to 40 C.F.R. § 1502.22(b). The FEIS contains such an analysis in Appendix F. While the regulations do not specifically state that a worst case analysis is subject to the "understandability" standard of § 1502.8, I find that it is. A worst case analysis is an integral and important facet of an EIS. It is designed to alert the public and the decisionmakers of potential health hazards and the probability of their occurrence. Unless the public and the decisionmakers are able to understand a worst case analysis, it is impossible for them to be aware of the potential risks.

In the present case, the worst case analysis contained in Appendix F of the FEIS is hypertechnical, complex and replete with lengthy equations and calculations. When asked to interpret a portion of the worst case analysis, defense witness Dr. Richard Wilson, Chairman of the Department of Physics at Harvard University, stated that he was unable to decipher the precise meaning of the passage, but given 15 minutes of study he could probably untangle the message. This testimony illustrates the complexity of this portion of the document.

I find that the worst case analysis does not meet the requirements of § 1502.8. Although the worst case analysis contains all the necessary information, it fails to communicate that to the persons entitled to be so informed. "An environmental impact statement is more than a disclosure document." 40 C.F.R. § 1502.1. An EIS must translate technical data into terms that render it an effective disclosure of the environmental impacts of a proposed project to *all* of its intended readership. *Natural Resources Defense Council v. United States Nuclear Regulatory Commission,* 685 F.2d 459, 487 n. 149 (D.C.Cir.1982), rev'd on other grounds sub nom. *Baltimore Gas & Electric Co. v. NRDC,* 462 U.S. 87, 103 S.Ct. 2246, 76 L.Ed.2d 437 (1983). *See also, Warm Springs Dam Task Force v. Gribble,* 378 F.Supp. 240, 252 (N.D.Cal.1974) (main text of EIS clear, concise and easily readable). *Sierra Club v. Froehlke,* 359 F.Supp. 1289, 1343 n. 215 (S.D.Tex.1973) rev'd on other grounds sub. nom. *Sierra Club v. Callaway,* 499 F.2d 982 (5th Cir. 1982) (federal agencies must screen EIS for words not understandable to average person).

Therefore, the worst case analysis is legally inadequate and cannot be relied upon for the spraying of the four synthetic pesticides. The other two alternatives, B.t. and

integrated pest management, are not covered in the worst case analysis, nor need they be. The use of these alternatives is fully covered in the main text of the FEIS, which I have determined legally adequate.

E. *Remedies*

The main text of the FEIS is legally sufficient, but the worst case analysis is legally insufficient. I now must determine the appropriate remedy. An adequate worst case analysis is required before any of the four synthetic pesticides can be used in the eradication or suppression of gypsy moths. I therefore enjoin the use of the synthetic pesticides carbaryl, trichlorofon, acephate and diflubenzuron. I do, however, delay the injunction for two reasons.

At close of trial I asked plaintiffs specifically what relief they sought, should I determine the FEIS inadequate. Plaintiffs requested an injunction in Oregon, commencing immediately, prohibiting the use of the four synthetic pesticides. They also asked for a nationwide injunction to commence January 1986.[1] I grant their request.

The Oregon spraying program has been approved and the federal government has agreed to contribute dollars for its implementation. That program does not envision the use of chemicals. Rather, it has long been decided that B.t. will be used. I have earlier ordered that the Oregon program can, therefore, proceed as planned.

I impose a delayed *nationwide* injunction because it is the plaintiffs' requested remedy and because the imposition of an injunction on the eve of national spraying of gypsy moths would result in more damage to the environment than allowing the use of the chemical spraying. The Ninth Circuit has firmly stated that an injunction may be modified or denied altogether if the court determines more harm to the environment would result with the imposition of the injunction. *American Motorcyclist Association v. Watt,* 714 F.2d 962 (9th Cir.1983). *See also, Alpine Lake Protection Society v. Schlapfer,* 518 F.2d 1089 (9th Cir.1975). The FEIS makes it clear that many states are heavily infested with gypsy moths and the use of the synthetic pesticides is necessary to control these infestations. If not challenged, the moths would destroy the foliage on millions of acres of hardwood forests in the eastern United States. Although B.t. is the treatment of choice in Oregon, *see* FEIS pages 3, 10, we do not know that it is elsewhere, and we do not even know if B.t. is available elsewhere. Therefore, the nationwide injunction will go into effect following the spraying season, which, on balance, seems the soundest decision in aid of a healthy environment.

CONCLUSION

[3] The main text of the FEIS is legally adequate, but the worst case analysis fails to meet the requirements of 40 C.F.R. § 1502.8 and is inadequate. For the reasons above stated, I enjoin the use of carbaryl, trichlorofon, acephate and diflubenzuron commencing immediately in Oregon, and after January 1, 1986 on a nationwide basis.

Narrative Statement of John Neisess

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IN THE UNITED STATES DISTRICT COURT

FOR THE DISTRICT OF OREGON

OREGON ENVIRONMENTAL COUNCIL; CITIZENS FOR THE SAFE CONTROL OF THE GYPSY MOTH; ELAINE OLSEN and GLEN OLSEN,

Plaintiffs,

and

FRIENDS OF THE EARTH; NATIONAL COALITION AGAINST THE MISUSE OF PESTICIDES,

Plaintiff-Intervenors,

v.

LEONARD KUNZMAN, Director, State of Oregon, Department of Agriculture; et al.

Defendants.

CIVIL NO. 82-504-RE

NARRATIVE WITNESS STATEMENT OF JOHN NEISESS

QUALIFICATIONS AND BACKGROUND

I, John Neisess, am a chemist currently employed as a pesticide specialist by USDA-Forest Service on the Washington Office's-Forest Pest Management staff. My major responsibility is to serve as the Forest Service coordinator for USDA's National Agricultural Pesticide Impact Assessment Program (NAPIAP). Under this program the Forest Service sponsors research to fill data gaps in information needed in EPA's reregistration process. Each year, since the program began in 1977, we have funded at least a dozen studies on subjects such as exposure, environmental fate, and economic benefits of pesticides currently registered for use in forestry. As a member of an inter-departmental Technical Advisory Group for NAPIAP I have reviewed many pesticide position documents written by EPA. Because of this experience gained from reviewing risk analyses written by EPA, the coordination of the NAPIAP, data gap research, and my previous research experience dealing with pesticides, I was asked to assist our Morgantown Field Office in the preparation of the risk analysis that was to be included in the 1984 and 1985 environmental impact statements.

Prior to my transfer to the Washington office in the fall of 1979, I worked for 9 years as a research chemist for the Pacific Northwest Forest and Range Experiment Station on the development of chemical and biological insecticides that could be used in forestry. As my attached curriculum vitae indicates, this research included work with Bacillus thuringiensis, Douglas-fir tussock moth virus, acephate, carbaryl, trichlorfon, diflubenzuron, mexacarbate, and natural and synthetic pyrethroids. In my research, I studied factors that influenced aerial application and efficacy. My publications in this area are listed in the attached curriculum vitae.

I received a Ph.D. in physical chemistry from Oregon State University in 1971. Over the past two years, I have received some training in toxicology through attendance at toxicology short courses. I participated in the development of a toxicology short course for Forest Service personnel with the cooperation of the American Chemical Society. The first class was held February, 1985.

TESTIMONY

Use of NOELs and ADIs. Our review of the scientific literature focused on determining the range of doses tested, what dose caused no effect, the lowest dose that caused an effect, and the effects observed. In all cases these were the observations of the authors of the research papers and did not require an interpretation by us. Where different studies reported different no observable effect levels (NOELs) for the same test animal, we used the lowest value so that we would not underestimate risks.

Since acceptable daily intakes (ADIs) are established by toxicologists at EPA after reviewing all available data, we felt that human exposure estimates could be compared to the ADIs as a first step in assessing risk to humans. If

exposure was below the ADI, we concluded that it was in an acceptable range. If the exposures exceeded the ADI as was the case with many of the worst case exposures, then NOELs were examined to see what possible adverse effect might occur. These effects could be broken down into such things as cholinesterase depression, methemoglobin formation, swelling of the kidney or liver, or birth defects, depending on the effect and NOEL reported in research studies. For example, the NOEL for cholinesterase depression in rats treated with acephate is 0.25 mg/kg, while the NOEL for birth defects is 10 mg/kg. Obviously the estimated worst case acephate dose of 0.147 mg/kg (which was above the ADI) might cause a depression in cholinesterase activity if it were administered on a daily basis, but it would not be expected to cause a birth defect.

Through inadvertence, the systemic NOELs of carbaryl were not listed in Table 2 of Appendix F. However, the information is contained in both Schulze et al. (1979) and USEPA (1984e) referenced in Appendix F at F-109 and F-111, respectively, and briefly discussed on page F-67. The NOEL of 10 mg/kg/day referred to on page F-67 comes from a 2-year-feeding study in rats. The effects observed at the highest dose tested (20 mg/kg/day) were cloudy swelling of convoluted and loop tubules in the kidneys and cloudy swelling of the hepatic cords about the central vein in the liver. USEPA (1984e) also lists a NOEL of 10 mg/kg/day for cholinesterase inhibition. No NOELs for immunosuppression were considered in the FEIS, since EPA does not require such studies for registration. (Our toxicologists will address the limitations of immunotoxicology studies).

To ensure that the risk analysis used proper toxicological data and risk analysis procedures pursuant to current standards, we had the seven experts listed on page 81 of the FEIS review a working draft of the risk analysis and the Draft Supplement. We followed their suggestions on the use of NOELs, ADIs and the calculation of cancer risk.

Estimates of Human Exposure. As stated on page F-27, carbaryl was the only one of the four insecticides for which data was available for worker and residential exposure resulting from spraying forest lands. Exposure was determined by measuring the amount of 1-naphthol, a breakdown product of carbaryl, that was in the urine of workers or residents who lived in or near the treatment areas. The measurement of 1-naphthol in urine allows for a direct measurement of exposure from all routes (contact with the skin, respiration, and eating) because it measures the amount of chemical actually in the body. Human exposure for acephate, trichlorfon, and diflubenzuron was extrapolated from the carbaryl data base. Although this extrapolation imparts uncertainty into the exposure estimates for those three insecticides because it assumes similiar absorption and metabolism rates, such extrapolation is a standard practice used when specific exposure data is not available.

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Exposure to nearby residents resulting from drift of the insecticides is addressed in the FEIS at page F-31. Based on a 1984 paper by Witt (cited at F-113), insecticide residues measured 250 feet away from the spray block were estimated to be about 2/3 of those measured within the spray block. At 1/4 mile away from the treatment block, spray residues would be only 2 percent of those found within the treated areas. These multipliers were used to determine exposure to residents from spray drift (see F-32).

As discussed on page F-37 to F-45, potential dietary exposure comes from three sources: water, vegetables, or meat from domestic or game animals that have eaten vegetation containing spray residues. When residue and persistence data were available from research studies involving spray trials against gypsy moths, these data were used in the risk analysis rather than data from agricultural or other forest spraying. Residue and persistence data related to gypsy moth spraying was available for water and forest vegetation. It was not available for meat (insecticide residues in animals) or the type of vegetables that could be growing in people's gardens at the time of gypsy moth spraying.

Data on carbaryl residues in water reported by LaFleur (1976) were not included in the analysis because that study involved a soil drench of 22.7 lbs. carbaryl/acre; this application rate far exceeds the one lb./acre use of carbaryl for gypsy moth control. The study by Gibbs et al. (1984) would have been added to the section on environmental fate of carbaryl if it had been brought to our attention during the comment period. Nevertheless, it is questionable whether the study has relevance to human health risk from drinking water in the gypsy moth program since Gibbs et al. reports the persistence of carbaryl in ponds in Maine. The relevance is questioned because the risk analysis concerns risks associated with drinking water, but chemical insecticides are not used to control gypsy moths around major water impoundments. Since people normally take drinking water from running water, this type of data was relied on in the risk analysis. However, the persistence information of Gibbs would become important if a specific project considered the use of carbaryl around a large water impoundment that was used as a source of drinking water. This is the type of information that should be more fully discussed in a site specific EA. Then, other factors affecting persistence such as pH, temperature, or sediment could also be taken into consideration.

It is important to note that insecticide residues on forest vegetation (such as maple leaves) were only used to calculate exposure to animals. Calculated exposures to animals resulted in worst case residue levels in meats that were 100 to 1000 times higher than any residue amounts actually measured from direct feeding studies (see pages F-37-38). Since these calculated worst case residue values are so much higher than those actually measured, use of any higher residue value on foliage would merely result in trivializing the worst case analysis.

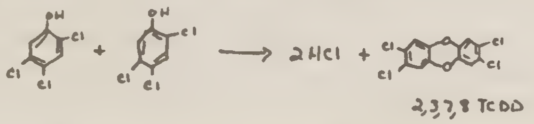
As noted on pages F-42 and F-43, residue data resulting from agricultural application to vegetables were used as a surrogate for residues that could be on vegetables as a result of aerial spraying for gypsy moth control. In my opinion, this surrogate should overestimate exposure, and therefore risk, because agricultural applications are generally made using ground equipment to release spray a few feet above the crop, thus maximizing deposit.

In the calculations of average lifetime exposure used to determine carcinogenic risk (see pages F-25, F-77, and F-80), the dietary components (water, meat, and vegetables) were lumped together. The residues were assumed to all degrade at the same rate. The slowest degradation rate was used for all media, and degradation was assumed to follow a linear course instead of the normal first-order reaction kinetics where there is usually a rapid initial loss of insecticide residue shortly after application. This type of lumping and degradation should overestimate exposure.

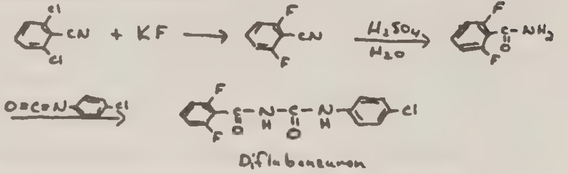
Possible Dioxin Contamination in Diflubenzuron. The concern that diflubenzuron might be contaminated with dioxin arose when an EPA internal memo was published in the February 20 issue of Pesticide & Toxic Chemical News. The Department of Agriculture contacted both the registrant and EPA when this issue came to its attention.

According to EPA (see attached EPA letter from Judith W. Wheeler to John Neisess dated March 21, 1985), the purpose for the list of pesticides "possibly contaminated with dioxin" was to identify potential candidates for analysis for possible halogenated dibenzo-p-dioxins or halogenated dibenzofurans contaminants at a 0.1 percent level of detection. EPA has indicated that the list was speculative and also concluded that it did not expect any 2,3,7,8-tetrachlorodibenzo-p-dioxin to occur in diflubenzuron. Moreover, the registrant has tested for possible presence of tetrachlorodibenzo-p-dioxins and tetrachlorodibenzofurans in technical grade diflubenzuron down to a level of 0.01 ppm. No contaminants were found at that level of sensitivity (see attached Uniroyal letter from C.A.Schadbolt to John Neisess dated March 20, 1985). It is important to note that this level of testing is at least 10 times more sensitive than what EPA is considering.

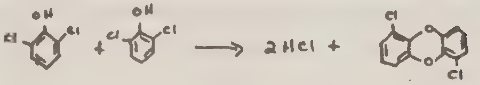
An understanding of chemical structure alone should put this issue to rest. The "dioxin" which is the object of public concern is 2,3,7,8,-tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD), the highly toxic contaminant present in the herbicide 2,4,5-T. The dioxin 2,3,7,8-TCDD is generally regarded as the most toxic of the 75 possible compounds in the class of chemicals known as chlorodibenzo-p-dioxins, whose toxicities widely differ. Both the number of chlorines and their position on the dibenzo-p-dioxin rings dictate the chemistry and therefore the toxicity of these compounds. The 2,3,7,8-TCDD in 2,4,5-T is formed because of the presence of 2,4,5-trichlorophenol. Simply stated, two chlorophenols react with the loss of HCl to form 2,3,7,8-TCDD:



Obviously trichlorophenol has to be present to form 2,3,7,8-TCDD. In fact, ortho-chlorophenol (the OH and Cl adjacent to each other on the benzene ring) must be present to form dioxins. Diflubenzuron is made by reacting dichlobenil with potassium fluoride to form 2,6-difluorobenzonitrile. This compound is hydrolysed to form 2-6-difluorobenzamide which is then reacted with 4-chlorophenylisocyanate:



Obviously no 2,3,5-trichlorophenol or any chlorophenol is present in this synthesis. A diagram of the synthesis demonstrates that the only place an ortho-chlorophenol could be present would be in the preparation of dichlobenil. This compound is also on the EPA list of "possibles" and is probably why diflubenzuron is on the list. If a chloro substituted phenol were an impurity of dichlobenil, a possible dichlorodibenzodioxin could form.



From this analysis of chemical structures of possible contaminants, EPA's conclusion that 2,3,7,8-TCDD would not be present in diflubenzuron and that the possibility of any dioxin was only speculative, and the fact that the registrant has analyzed for dioxin contaminants to levels below 0.01 ppm, I concluded that the issue of possible dioxin contamination was not a substantive issue. I advised our decision makers of these findings and recommended that possibility of dioxin contamination was not a significant new issue requiring a supplement to the FEIS.

Cancer Risk. As stated on page F-14, carbaryl was considered non-carcinogenic. This statement is based largely on the review of 10 different studies by EPA in the Carbaryl Decision Document (USEPA 1980a). Although some of the studies were considered flawed because of poor experimental design or reporting, collectively those studies provide sufficient evidence that carbaryl is not oncogenic in laboratory animals. EPA reiterated this finding by not requiring additional oncogencity studies in the carbaryl registration standard issued March 30, 1984 (USEPA 1984a).

Regarding the cancer-potency of N-nitrosocarbaryl, the paper by Lijinsky and Taylor (1976) does not discuss the use of a control in the methods and materials section. The footnote merely states: "None of these tumors was (sic) seen in untreated control Sprague-Dawley rats of our colony." The paper does not provide the reader with any information which indicates the use of two control groups, as is customary with gavage studies. One is a control group treated by the gavage method, intubation to the

stomach, but without the test substance. The other is an absolute control group which has no treatment at all.

I have since discussed the studies by Eisenbrand et al, and Lijinsky and others with Dr. Gio Batta Gori, one of our consultants specializing in cancer (see attached letter from Dr. Gori to John Neisess dated April 1, 1985). Dr. Gori pointed out that the Eisenbrand et al. (1976) study may have also lacked two controls. A later study by Lijinsky and Schmahl (1978), which was not brought to our attention during the comment period, did contain both controls. Dr. Gori also pointed out that the gavage method of testing was reviewed by an Ad Hoc Panel on Chemical Carcinogenesis Testing and Evaluation of the Natural Toxicology Program chaired by Dr. John Doull. That panel was generally critical of the gavage method because it introduces confounding variables in toxicity tests and therefore may overestimate the incidences of cancer in the forestomach.

If the cancer incidence data reported in Lijinsky and Taylor (1976) were used to calculate the cancer potency of N-nitrosocarbaryl in humans, the β terms would range from 4.6 to 10.5 $(mg/kg/day)^{-1}$ if the controls were assumed to be zero. If incidence data from Lijinsky and Schmahl (1978) were used, the cancer potency in humans would range from 0.25 to 0.67 $(mg/kg/day)^{-1}$, because higher doses were used in that study. This 10-fold inverse difference in dose response cannot be explained from information that is presented in the two publications. The difference between the 1976 Eisenbrand study (β= 0.057

$(mg/kg/day)^{-1}$) and the Lijinsky studies can be explained by the acute toxicity seen at maximum doses in the Eisenbrand study. Using cancer potency values from the Lijinsky studies, the weighted lifetime risk of cancer would range from 1.5×10^{-8} to 6×10^{-7} for an individual exposed to carbaryl during suppression projects (compared to 3.5×10^{-9} currently used in the FEIS). The important point is that, given the uncertainty in cancer potency of N-nitrosocarbaryl, the worst case risk is still below 1×10^{-6}, or less than one in a million. This worst case risk, or any lower risk, can only be realized if N-nitrosocarbaryl forms in humans. Direct administration of carbaryl and nitrite given to rats by gavage failed to produce tumors of the forestomach (Lijinsky and Taylor 1977). This result reemphasized the fact that although N-nitrosocarbaryl is a carcinogen, the real uncertainty is whether it will form in the stomach of humans.

Trichlorfon. The question about adequacy of the cancer data for trichlorfon was first raised during the public comment period by Mr. Kenneth Hobbs (comment letter 13g). An inspection of the data used to calculate the cancer potency for trichlorfon (F-19) shows that only 4% of the rats treated with the highest dose (1000 ppm in diet) had tumors, while 7.5% of the untreated control rats had tumors. Obviously the treatment did not cause a statistically significant increase in tumors. We were forced to assume that the untreated control group actually had zero tumors in order to estimate an upper bound of cancer risk. As noted in Comment Response 13 g (G-18), this constituted

a worst case assumption where uncertainty existed about the carcinogenicity of trichlorfon. EPA concluded that our assumption that trichlorfon was a "...carcinogen represents a prudent approach to analyzing the potential risk associated with trichlorfon's use." (Comment Letter 29 d).

Diflubenzuron. Based upon the recently completed cancer bioassays of diflubenzuron, EPA advised us that metabolites of diflubenzuron (such as 4-chloroaniline) do not pose a cancer risk to humans through dietary exposure (see attached EPA letter from Allan Hirsh to James Stewart dated March 8, 1985, A.R. 8c). EPA reasoned that the diet used in rat and mouse cancer studies would contain not only diflubenzuron but also the metabolite, 4-chloroaniline, because the diflubenzuron would be degrading. The animals themselves would also metabolize diflubenzuron into 4-chloroaniline and thus receive additional exposure to the metabolite. The cancer studies therefore measured cancer risk associated with levels of 4-chloroaniline that might be associated with the diet.

The NCAP comment letter (letter 12) prompted us to take a second look at data concerning 4-chloroanaline. We concluded that where diflubenzuron was metabolized by an animal, there might be a possibility of proportionately higher levels of 4-chloroanaline in animal tissue than would have resulted from the rat diet. This was certainly true for fish. As stated on page F-83, data were not available on 4-chloroanaline levels in meats other than fish. Residues in fish were considered to be the worst case because 60% of the metabolic residues in fish were 4-chloroaniline, which was the highest metabolic rate in animals reviewed by EPA (USEPA 1979). Other dietary components such as water or vegetables were not considered in the analysis because we felt the 4-chloroaniline levels would be similiar to those in the rats diet. In other words we only looked at additional risks associated with 4-chloroaniline residues that could occur as a result of diflubenzuron being metabolized to levels higher than what would have been tested in the cancer bioassays using diflubenzuron.

Since completing the FEIS, we have acquired a copy of the NCI study "Bioassay of p-chloroaniline for possible carcinogenicity" (AR 12a). Dietary concentrations of 4-chloroaniline were 0, 250, and 500 ppm for rats, and 0, 2500, and 5000 ppm in mice. The only neoplastic lesions found that were considered as possibly being related to the 4-chloroaniline treatment were fibromas and sarcomas in the spleen of male rats and hemangiomatous tumors in mice. In both cases the incidences of these tumors were not statistically significantly greater than those in control animals.

Incidences of tumors in mice were 2 out of 20 control males, 10 out of 50 low dose males, 14 out of 50 high does males, 0 out of 18 control females, 3 out of 49 low dose females, and 8 out of 42 high dose females. Using the data for males (because of the greater incidence rate), cancer potency for humans would be 0.0037 $(mg/kg/day)^{-1}$. A cancer potency can also be calculated

from the combined incidence of splenic fibromas, fibrosarcomas, hemangiosarcomas, osteosarcomas and sarcoma NOS in male rats of 1/20 controls, 0/49 low dose, and 10/49 high dose. In this case the cancer potency for humans will be 0.035$(mg/kg/day)^{-1}$.

Comparing these cancer potency values to the value of 0.025$(mg/kg/day)^{-1}$ used in the discussion of risk on page F-83 shows that risks were either underestimated by a factor of 1.4 or overestimated by a factor of 6.8 depending on whether the rat or mouse data were used to estimate cancer potency. These variations in risk are small compared to the uncertainty in possible human exposure that required the assumption that 4-chloroaniline residues may persist in meat for 60 days, although the Diflubenzuron Decision Document (USEPA 1979) stated that residues are rapidly depleted. Because the exposures are over-estimated (because of the 60 day persistence), I believe it is safe to say that the cancer risk associated with 4-chloroaniline is in the 1 X 10^{-8} range.

Synergism. The 10-fold synergistic increase in toxicity or risk was discussed only for the four chemical insecticides included in the FEIS. To consider the synergistic effect of just one of these insecticides on the toxicity of all chemicals that could be in the environment is an unrealistic task. Since there are literally thousands of chemicals in the environment, this would have required a literature review of the toxicology of all possible chemicals. The worst case 10-fold increase in toxicity resulting from the synergistic effect would then have to be applied to each and every chemical. In other words, a risk analysis would need to be completed for each chemical that could be in the environment.

As noted on page F-102, EPA stated that they had examined considerable data and found no evidence of potentiation. For example, carbaryl was tested in combination with 47 other pesticides, but potentiation was noted in only one case. The concentration of the combined chemicals is also important in any discussion of synergistic effect. In the studies by Statham and Lech (1975a, 1975b and 1976 cited at F-110), the carbaryl concentration was 1 ppm. Since this concentration is considerably higher than the highest carbaryl residue reported from forest spraying (Gibbs et al. 1984), it is uncertain if synergistic effects would occur at doses or concentrations resulting from spraying for gypsy moth control. Dr. Wilson will discuss further the effect of concentration as it relates to synergism.

CONCLUSION

I believe we have made a good faith effort in the FEIS to comply with NEPA and to provide USDA decision makers with the information necessary to identify the human health and environmental risks associated with using accphate, Bacillus thuringiensis, carbaryl, diflubenzuron, or trichlorfon to control gypsy moths. I want to empahais that our evaluation of pesticide risks is a dynamic process. For example, we currently have a contract with MITRE corporation to review the scientific literature on the toxicology and environmental fate of pesticides used in forestry

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(including those used in the gypsy moth control programs). Our NAPIAP Program will continue to fill gaps in the relevant information needed to evaluate risk and benefits of using pesticides in forestry.

John Neisess
JOHN NEISESS, Ph. D.
Pesticide Specialist
Forest Pest Management

Dated: April 4, 1985

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John Neisess, Ph.D.

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EDUCATION

B.A.	Central Washington State College, Ellensburg, WA - 1966
Ph.D.	Physical Chemistry, Oregon State University, Corvallis, OR - 1971

BACKGROUND

1979-Present	Pesticide Specialist, Forest Pest Management staff, USDA Forest Service, Washington DC. Forest Service Coordinator of National Agricultural Impact Assessment Program.
1975-1979	Research Chemist Research Work Unit - Disease of Western Forest Insects Pacific Northwest Forest and Range Experiment Station, Corvallis, Oregon
1971-1975	Research Chemist Research Work Unit - Aerial Application of Pesticides Pacific Northwest Forest and Range Experiment Station, Corvallis, Oregon

PROFESSIONAL SOCIETIES

(1) American Chemical Society
(2) Sigma Xi
(3) Entomological Society of America

HONORS AND AWARDS

USDA SUPERIOR SERVICE UNIT AWARD, 1977

PAPERS PRESENTED

(1) 1965. Ion transport through biological membranes. ACS, Student Affiliates Meeting, Ellensburg, Washington.

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UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

MAR 8 1985

OFFICE OF
EXTERNAL AFFAIRS

FOREST SERVICE
RECEIVED
MAR 11 1985
FOREST PEST
MANAGEMENT

Mr. James L. Stewart
Director, Forest Pest Management
U.S.D.A. Forest Service
P.O. Box 2417
Washington, D.C. 20250

Dear Mr. Stewart:

On February 27, 1985, Mr. John Neisess of your staff telephoned a member of my staff to request information and some background materials concerning the Environmental Protection Agency's (EPA) position on the potential health effects of the pesticide Dimilin (diflubenzuron). We understand that the request relates to the preparation of the Supplemental Environmental Impact Statement (EIS) for the Forest Service's proposed gypsy moth control program for 1985 and the associated court action and that therefore time is of the essence. Accordingly, we have obtained information from our Office of Pesticides Programs responding to the following specific requests:

(1) a copy of the document "Risk Analysis for Dimilin and Alternatives for Aquatic and Terrestrial Wildlife - Cotton, Soybeans, Hardwood Forests and Mosquito Control," Tucker and Stevens, EPA, 1978;

(2) information on the current Agency position concerning rat and mouse oncogenicity studies submitted by the registrant of Dimilin; and

(3) information concerning the Agency's position on metabolites of Dimilin.

Enclosed is a copy of the Tucker and Stevens document. It should be noted that we regard this document as outdated because newer and more conclusive studies on health effects of Dimilin are available for risk assessment purposes.

The Agency has just completed reviews of rat and mouse oncogenicity data on Dimilin submitted to EPA by the registrant, Duphar. Enclosed is a copy of our letter to the registrant stating our conclusions. As you will note, the data support the conclusion that Dimilin is not oncogenic to rats or mice at doses up to and including 10,000 parts per million in the diet. This finding significantly alleviates previous concerns about the use of Dimilin.

Since there is a dog feeding study still underway, the Agency is currently utilizing a provisional acceptable daily intake level (PADI) for Dimilin, as described in the enclosed letter. This PADI is calculated in reference to met-and sulfhemoglobin formation, which are reversible toxic effects on blood cells, not related to oncogenicity.

In relation to metabolites of Dimilin, the Agency's current position can be summarized as follows. Since the anticipated maximum theoretical dietary exposure to residues of Dimilin is only ca. 3 % of the Maximum Permissible Intake (MPI), we are not at this time requiring additional animal metabolism data. Overall, the Agency considers that there are sufficient general animal metabolism data to support continued registration of the product under current conditions of use. The Agency may reconsider this position should additional residues be added to the Theoretical Maximum Residue Contribution (TMRC) or should unanticipated adverse toxicity data become available.

The rat and mouse feeding studies described above are a further indication that metabolites of Dimilin are not an oncogenic hazard through dietary exposure.

I hope the information provided is helpful. If we may be of further assistance please let me know.

Sincerely yours,

Allan Hirsch

Allan Hirsch
Director
Office of Federal Activities

Enclosures

The Franklin Institute
Policy Analysis Center

April 1, 1985

Dr. John Neisses
U.S. Forest Service
P.O. Box 2417
Washington, D.C. 20013

Dear Dr. Neisses:

With regard to the potential carcinogenic risk to man of N-nitrosocarbaryl (NNC), one must consider first the significance of the published tests for carcinogenicity in animals, and second the likelihood of the formation of NNC in man.

For the first issue, I have reviewed references 1, 2, 4, 6 and 7. Without exception all these studies are designed as exploratory research efforts and their conclusions are not acceptable according to formal bioassay guidelines. Neither NNC nor carbaryl itself are listed in the latest official Third Annual Report on Carcinogens of the U.S. Department of Health and Human Services (10). In all experiments, the number of animals is too small, control animals are either lacking or deficient, doses are selected without a biologic or metabolic rationale, dosage schedules are equally empirical, and weight variations in time are not reported. The outcomes are predictably erratic as the following table summarizes.

Reference	Total Dose mg/kg	# Treat-ments/# Weeks	Species Sex	Vehicle	% Animals with Carcinoma
2	5000	40/20	SD - M	olive oil	29
1	1500	1/1	W - M+F	starch	0
4	1500	40/20	SD - M	olive oil	47
7*	1200	100/50	Swi - F	acetone	40
1	1000	1/1	W - M+F	starch	0
6	600	10/10	SD - M	corn oil	21
6	600	10/10	SD - F	corn oil	57
4	500	10/10	SD - F	olive oil	75
4*	220	40/20	Swi - F	acetone	0

Note: SD = Sprague-Dawley rats. W = Wistar rats. Swi = Swiss mice. References marked * used the skin painting route, all others used gastric intubation (gavage). Total doses are best approximations from the data reported, and assume an average weight of 100 g for female rats, 200 g for male rats, and 20 g for mice.

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The experiments using a single gavage dose in a starch paste vehicle were probably inadequate to stimulate initiation, which seems to require a longer lasting presence of the agent. Similar considerations are probably valid for the skin painting experiment at 220 mg/kg total dose. The other experiments give the impression of an overall inverse dose response, especially if one excludes the male SD rats in reference 6. From the survival data reported in most studies, these anomalous results are likely the consequences of the marked toxicity of NNC and the improper selection of dose regimens.

A remarkable finding is that carcinomas all originate at the site of application and only there, indicating that the agent is either unstable or is metabolized very rapidly to innocuous by-products. Moreover, all gavage experiments are subject to the criticism raised against this route of administration by the National Toxicology Program Ad Hoc Panel on Chemical Carcinogenesis Testing and Evaluation, chaired by Prof. John Doull (8), which culminates years of concern on the part of cautious scientists who recognize the un-natural conditions of a modality that traumatizes the animals, distorts the distribution and absorption of the dose, and affects its metabolism and disposition beyond recognizable extremes of normalcy.

All these considerations reinforce the notion that we are dealing with an agent that is effective in the species tested only topically and at very high doses. In view of this, it may be improper to declare total dose in terms of mg/kg of body weight, when in fact one should speak of effective dose/cm², calculated with due regard to frequency of application, duration of treatment, and surface area of tissue treated. Such computation would have to depend on some elastic assumptions and is beyond the scope of this present review. However, and for instance, assuming that one application a week per 10 weeks does not amplify general toxicity and is sufficient for initiation and that the forestomach has a surface of 1 cm², then the most recent experiment (6) indicates that 10 applications over a 10 week period at 120 mg/cm² on the forestomach of male SD rats results in 21% incidence of carcinomas, while for the females a total dose of some 60 mg/cm² results in a 57% incidence. These inferences of course are still speculative, yet they reflect the observations more realistically than the total dose expressed in mg/kg of body weight.

Regarding the issue of the possible formation of NNC in man, one must consider that even after many in vitro facilitations are provided, the reaction of carbaryl with nitrite is very inefficient (1,5). At extremely high concentrations of 0.5 M carbaryl and 2 M nitrite a conversion of only 5.7% was obtained in HCl, and only after the addition of 25% DMSO to solubilize carbaryl (5). Notice that a 0.5 M solution requires approximately 105 g/liter of carbaryl. In a more reasonable experiment (1), a solution of 0.001 M carbaryl obtained a 1.7% yield at

37°C for one hour in 0.1 N HCl. On the other hand, well confirmed studies report that ascorbic acid (vitamin C), a normal component of human diet, is an effective inhibitor of the nitrosation reaction (11).

In fact, the direct administration of carbaryl and nitrite to rats failed to result in any tumor increase in chronic transplacental bioassays (5). For man, there is no published or known evidence of increased cancer experience in workers with the highest exposures to carbaryl, either because such doses are below an effective threshold, or because man does not form significant NNC levels in the stomach and metabolizes or neutralizes very rapidly whatever is formed, or because man lacks a specific target tissue such as the forestomach of the rat. In fact, it is evident that the glandular stomach of the rat -- the reasonable counterpart of the human stomach -- is not affected by NNC despite the spillage of active agent that must occur after dosing the forestomach by gavage.

In conclusion, all the authors of the studies reviewed also note in several occasions, NNC appears to have a carcinogenic affinity exclusive for the forestomach of the rat and, in one isolated experiment, for mouse skin. The published instances where carcinogenic effects were not apparent, suggest that NNC is a poor initiator, requiring frequent and prolonged contact with the target tissue at very high concentrations per unit area of tissue surface (1,4,5).

Considering the low levels and frequency of possible human exposure to the combination of carbaryl and nitrites, the extremely low yields of the nitrosation reactions in the stomach, the protective function of dietary ascorbic acid and the absence in man of a target tissue corresponding to the rat forestomach, it is reasonable to conclude that the probability of carcinogenicity in man due to NNC is extremely remote.

How remote, is a conjecture that is objectively unresolvable, and in approaching quantitative assessment of human risk based on the available animal data for NNC, it is necessary to keep in mind the uncertain significance of any such exercise, a precaution that has been officially recognized (9,13). If some arbitrary estimate is required notwithstanding all reasonable caveats, then an extrapolation based on effective dose per unit of tissue surface area will be more appropriate than computations based on a total dose per kg of body weight. Because the animal results are also affected by frequency and total time of application it will be also necessary to tranpose such kinetics into human equivalent parameters: an exercise that surely will compound the speculative character of any outcome. Under the two scenarios, and setting aside for sake of simplicity all considerations of dosing frequency, the computation of total dose in the experiment of reference 6 will be 600 mg/kg body weight or 120 mg/cm² in male SD rats, as mentioned above. For a 70 kg person with a 18 X 10^3 cm^2 surface (12) the equivalent doses would be 4.2 grams on a body weight basis assuming any route of entry, and 2160 grams on a body surface basis, assuming an even dispersion of NNC in the environment so that at least 120 mg are applied to 1 cm^2 of surface. In other words, the concentration of NNC in environmental media (air, water, etc.) would have to be over 500 times higher in order to provide equivalent surface activity levels.

It follows that a human cancer risk assessment exercise based on total dose per kg body weight will provide margins of prudence that are substantial and should amount to several hundred-fold overstatements.

With all good wishes,

Gio B. Gori

Gio B. Gori, DSc, MPH, DATS

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March 20, 1985

Dr. John Niesess
USDA Forest Service
Forest Pest Management
Rm. 204 RTD
1611 N. Kent St.
Roslyn, VA 22209

Dear Dr. Niesess:

With reference to the issue of possible contamination of diflubenzuron by the presence of dioxins, we have received information from Duphar B.V. of the Netherlands, the manufacturer of the product, which should allay all fears of such contamination.

In 1982-1983, the matter of possible presence of dioxins was studied by Duphar. In their analytical studies on the possible presence of tetrachlorodibenzodioxins and tetrachlorodibenzofurans in technical diflubenzuron, the limit of detection was 0.01 part per million, which is 10 to 100 times better than the range stated in Pesticide & Toxic Chemical News, the apparent source of the concern. In this phrasing, tetrachlorodibenzodioxins represents a total of 22 possible isomers of this compound. Similarly, tetrachlorodibenzofurans represents a total of 38 possible isomers. Neither tetrachlorodibenzodioxins nor tetrachlorodibenzofurans are present in technical diflubenzuron.

Since for practical purposes we are talking about DIMILIN WP 25%, a dilution factor of 4 can be applied. Arithmetically, this brings down the detection limit to 0.0025 ppm.

With regard to the possibility that phenolic compounds used in manufacture could lead to the presence of dioxin, the manufacturing process of diflubenzuron does not use parachlorophenol or any other chlorinated phenolic substance. Therefore, mono-, di-, tri-, or polychlorinated phenols are not present in technical diflubenzuron.

We trust this will provide the information requested.

Sincerely yours,

CHEMICAL GROUP
UNIROYAL, INC.

C. A. Shadbolt
C. A. Shadbolt
Technical Manager, Insecticides
Crop Protection
Research & Development

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UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

March 21, 1985

OFFICE OF
GENERAL COUNSEL

John Neisess
U. S. Department of Agriculture
U. S. Forest Service, Forest Pest Management
Room 204 RPD
1611 North Kent
Rosslyn, VA 22209

Dear Mr. Neisess:

In response to your recent inquiries to the Environmental Protection Agency concerning the inclusion of diflubenzuron in a list of chemicals possibly contaminated with halogenated dibenzo-p-dioxins, I am enclosing a memorandum which I believe is responsive. If you have any further questions, please feel free to contact me.

Sincerely,

Judith W. Wheeler

Judith W. Wheeler
Attorney
Pesticides & Toxic Substances
Division (LE-132P)

Enclosure

cc: Dorothy Burakries
U. S. Department of Justice

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MANAGEMENT

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

MAR 21 1985

OFFICE OF
PESTICIDES AND TOXIC SUBSTANCES

MEMORANDUM

SUBJECT: Inclusion of Diflubenzuron in the "List of Chemicals Possibly Contaminated with Halogenated Dibenzo-p-dioxins."

FROM: Robert Brown, Deputy Director
Registration Division (TS-767) Robert W. Brown

TO: Judith W. Wheeler, Attorney
Pesticides and Toxic Substances Division (LE-132P)
Office of General Counsel

I am providing the following information in response to your inquiry concerning the inclusion of diflubenzuron (Dimilin) in the list of chemicals possibly contaminated with halogenated dibenzo-p-dioxins (HDDs) prepared by the Office of Pesticide Programs (OPP) in January, 1985, for internal program use. This list was developed for use in our planned data call-in effort to update Confidential Statements of Formula for currently registered pesticide products and to be used in response to a petition filed with the Agency by the National Wildlife Federation and the Environmental Defense Fund (NWF/EDF) concerning the regulation of HDDs and halogenated dibenzofurans (HDFs).

The purpose of the OPP list was to identify pesticide chemical candidates for which the Agency might request analyses for HDD or HDF contaminants at levels less than 0.1%, which is the level at or above which the Agency currently requires manufacturers to report contaminants in pesticides. The list is speculative and, as was noted in the memorandum accompanying the list, neither manufacturing processes nor Confidential Statements of Formula were considered in developing the list. Thus, the list is theoretical only, and not based on hard evidence. It was intended to encompass all pesticides which might contain any levels of any HDDs or HDFs. The classification scheme developed by the Agency, described in the monograph "Dioxins" by M. P. Esposito, T. O. Tiernan, and F. E. Dryden, document number EPA-600/2-80-197, November, 1980 (p. 37) was used as a rough guide in choosing the criteria for selection, but a number of pesticides included in this OPP list were not included in the "Dioxins" monograph. Selection criteria used for the OPP list were described in the cover memo.

In regard to diflubenzuron, the likelihood of it containing dioxin is extremely remote. Any HDDs or HDFs formed would most likely be a result of reactions between impurities in the starting materials, or intermediate reaction products. Diflubenzuron is only distantly related to structures known to form HDDs or HDFs; it is not an ortho-chlorophenol, which is the group of chemicals known to form dioxins. The HDDs and HDFs, if any, which might theoretically be formed would be those with lesser degrees of halogenation; we would not expect any 2,3,7,8-TCDD to occur. Diflubenzuron thus falls into Class III of the "Dioxins" criteria, as possibly but not likely containing HDDs and HDFs.

cc: Dioxin S. F.
Reading File
Diflubenzuron S. F.
Anne Barton, HED
Amy Rispin, HED
Chuck Trichilo, HED
L. Bradley, HED
Gunter Zweig, RD
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